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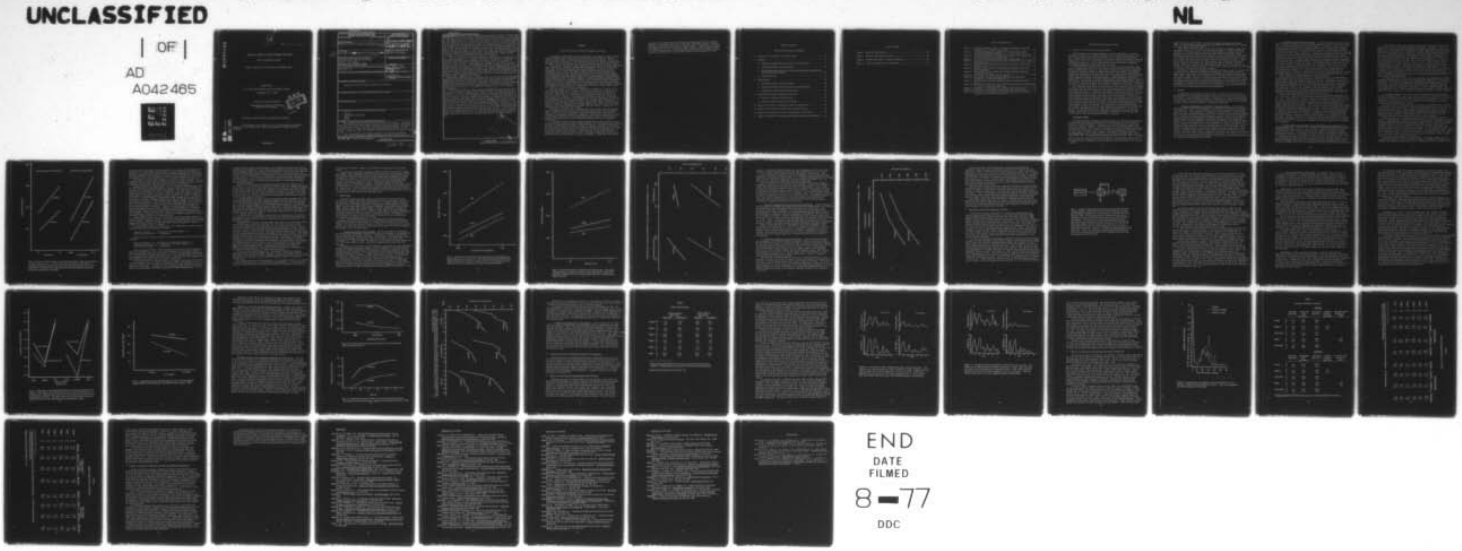

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EFFECTS OF ALCOHOL ON HUMAN PERFORMANCE AND SLEEP

Final Comprehensive Report

Boyd K. Lester, M.D., and Harold L. Williams, Ph.D.

Supported by

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Two separate investigations were supported by this contract. The principal aim of the first project was to analyze the effects of alcohol and certain commonly abused drugs on information processing and verbal memory. The aim of the second was to examine the acute and chronic effects of alcohol on human sleep. Progress on these two projects is reviewed in separate sections of this report.

Over the past few years we have organized a variety of experimental procedures into a systematic, sequential program for analyzing the effects of

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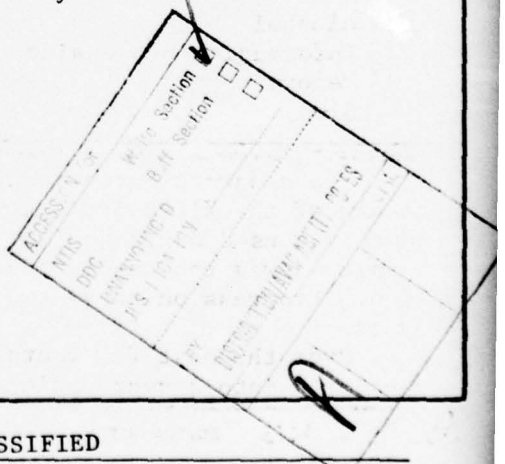
20. Abstract

alcohol and other drugs on human information processing and memory. At the core of this program is a set of basic experiments, each of which overlaps the others to some degree while, at the same time, providing a focus on a particular aspect of information processing. Selected on the basis of a series on pilot studies, these experiments were designed to examine the following aspects of performance: (1) the ability of the intoxicated subject to perform previously acquired routine tasks; (2) his ability to register, encode, learn, and retain verbal information; and (3) his ability to interrogate and retrieve information from his own short- and long-term memory systems. These core experiments were also designed to sample information processing operations ranging from the initial sensory registration of a stimulus to the execution of a response.

Once the effects of a drug were tentatively localized to a general set of processing operations (e.g., response selection and execution), a second set of converging experiments was performed to focus more clearly on the impaired function. Thus, there was a progression of experiments, each selected on the basis of previous results. This progression provided a number of constructive replications of our research. Such replications not only strengthen the validity of the findings, but also their generality. For example, when such diverse experiments as reaction time (RT) to visual digits, free recall of English nouns, and errors made in copying auditory letters indicate that a drug such as alcohol affects a particular information processing stage (see below), a great deal of confidence may be placed in the results.

In general, ~~our~~ the results are consistent with the view that alcohol disrupts cognitive operations associated with information output rather than information input. That is to say, the ability of the intoxicated person to encode and categorize information is relatively unimpaired. His deficits seem to be located in operations associated with the organization and retrieval of information in memory and with selection of the appropriate response from a set of response alternatives.

In our sleep studies we first compared physiological sleep profiles in sober chronic alcoholics and age-matched normal controls. We then examined the effects of two days of drinking on the sleep of chronic alcoholics. Analyses of EEG sleep data from sober alcoholics and control subjects confirmed that the amount of stage 4 sleep was significantly reduced in alcoholics, the deficit being most apparent in patients under 40 years of age. A major finding was that this disparity between alcoholics and controls was due to a decrease in amplitude of delta EEG rhythms rather than disappearance of delta frequencies. Along with loss of EEG amplitude, the sleep of sober alcoholics was characterized by more frequent arousal, accelerated periodicity of stage REM, increased autonomic activity and higher respiration rates than in normal controls. The effects of two days of drinking on the sleep of alcoholics were similar to those reported earlier for normal subjects; i.e., initial sedation, potentiation of slow-wave sleep, reduction of non-specific electrodermal activity and increased heart rate.



Summary

Effects of Alcohol on Human Performance and Sleep

Two separate investigations were supported by this contract. The principal aim of the first project was to analyze the effects of alcohol and certain commonly abused drugs on information processing and verbal memory. The aim of the second was to examine the acute and chronic effects of alcohol on human sleep. Progress on these two projects is reviewed in separate sections of this report.

Over the past few years we have organized a variety of experimental procedures into a systematic, sequential program for analyzing the effects of alcohol and other drugs on human information processing and memory. At the core of this program is a set of basic experiments, each of which overlaps the others to some degree while, at the same time, providing a focus on a particular aspect of information processing. Selected on the basis of a series of pilot studies, these experiments were designed to examine the following aspects of performance: (1) the ability of the intoxicated subject to perform previously acquired routine tasks; (2) his ability to register, encode, learn, and retain verbal information; and (3) his ability to interrogate and retrieve information from his own short- and long-term memory systems. These core experiments were also designed to sample information processing operations ranging from the initial sensory registration of a stimulus to the execution of a response.

Once the effects of a drug were tentatively localized to a general set of processing operations (e.g., response selection and execution), a second set of converging experiments was performed to focus more clearly on the impaired function. Thus, there was a progression of experiments, each selected on the basis of previous results. This progression provided a number of constructive replications of our research. Such replications not only strengthen the validity of the findings, but also their generality. For example, when such diverse experiments as reaction time (RT) to visual digits, free recall of English nouns, and errors made in copying auditory letters indicate that a drug such as alcohol affects a particular information processing stage (see below), a great deal of confidence may be placed in the results.

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I. Isolation of Information Processing Stages

The information processing approach to assessing the effect of various psychoactive drugs is to consider the human S as a communication system. The total time required to process information in such a system may be divided into a number of discrete intervals, each of which reflects a different operation. Using such a model, one can undertake a systematic experimental analysis of the hypothesized operations, their relationships, and the experiment treatments (including psychoactive drugs) which influence them.

Sternberg(1969) recently proposed an information processing method using choice reaction time to focus on the changes observed between input and output. Given a hypothetical model of a communication system and several experimentally manipulated variables, whose use makes sense within the context of the model, Sternberg proposes that one can use the patterns of interactions and additivity between the treatments to validate the model. Treatments which produce statistically independent effects of RT performance (so that no positive, two-way or higher order interactions are found) are presumed to influence separate operations (or stages). In contrast, when two treatments influence a single stage, their effects on RT should usually be in the form of a positive interaction. Exceptions to these two rules are possible (see Sternberg, 1969) but are assumed to be rare.

If a pharmacological agent is introduced as a new treatment in the Sternberg procedure, then any positive interactions of the agent with established treatments would imply that the drug influences the stage(s) mediating the effects of the established treatments. Conversely, an additive relationship between a drug and an established treatment would imply that both the pharmacological agent and the known variable affect different information processing stages. The use of a pharmacological variable should also help confirm or disconfirm current information processing models by providing constructive replications of prior research. Such replications strengthen the validity of the model hypothesized, and increase its generality by showing that the model holds under altered experimental conditions (Sidman, 1960). For example, if the pattern of interactions between established treatments remains the same under the drug as in controls, then both the validity and generality of the model would be strengthened. If unusual patterns of interactions between established treatments occur under the drug, however, then either (1) the drug completely rearranges the way information is processed or (2) the hypothesized model is incorrect.

Sternberg's Model

The basic pardigm from which Sternberg develops his information processing model involves Ss interrogating memory* to decide whether or not a recently presented stimulus belongs to a memorized list and then making an appropriate positive or negative response. Sternberg's model for this task assumes that each S sequentially: (1) encodes the stimulus, (2) serially compares the encoded stimulus with each memory set item; (3) selects the appropriate response ("yes" or "no"), and (4) executes that response. Sternberg's data imply that the discriminability of the stimulus affects the "stimulus encoding stage" (Sternberg, 1967); that the size of the memorized list influences the "serial comparison

*No more than four sample stimuli are included in the memorized list, so the interrogated memory is probably a limited-capacity, short-term store (see, section C below).

stage" (Sternberg, 1966; 1967); and that the stimulus-response uncertainty (SRU) and the stimulus-response compatibility (SRC) both influence the "response selection stage" (Sternberg, 1969).

Other models using the same task or very similar ones generally agree with Sternberg except that a "stimulus categorization stage," in which familiarity is judged, is often assumed to occur after the stimulus is encoded, but before memory is interrogated (Briggs & Swanson, 1969; Clifton & Gutschera, 1971). Furthermore, when the stimuli are as complex as words, Atkinson and Juola (1973) and Seamon (1973) have found that there may be no memory search on some trials. As a result, they postulate that after a familiarity estimate is made, a search is required only if the amount of uncertainty is above some criterion set by the S. With low uncertainty, the memory search stage is skipped. Finally, Rabbitt (1971) has cautioned that the Sternberg model does not incorporate the concept of "speed-accuracy trade-off." As a result, payoff procedures are essential to maintain a constant level of this variable.

Sternberg's model has advantages over earlier systems in both its theoretical concepts and its associated experimental methodology. Furthermore, numerous constructive replications have supported or extended the model (Sternberg, 1966; 1967; Bracey, 1969; etc.), thus strengthening its validity within a limited experimental setting. Finally, it provides a procedure for determining the "locus of effect" within the model for new treatments (e.g., such as psychoactive drugs) by examining the pattern of interactions and additivity of the new factor with experimental treatments whose effects are already known. As a result, our core experiments to examine the effects of various drugs include several experiments used by Sternberg and generally employ his methodology.

A. Alcohol

According to the procedure outlined above, if alcohol is introduced as a new treatment in the Sternberg procedure, then any interactions of alcohol with established treatments would imply that the drug influences the stages mediating the effects of the established treatments. Conversely, an additive relationship between alcohol and an established treatment would imply that the drug and known treatment affect different stages of information processing.

1. Stimulus Compatibility and Stimulus Discriminability. In the first of three experiments with alcohol (Tharp et al., submitted to Psychopharmacologia; Williams et al., 1973), recorded alphabet letters were presented to intoxicated and placebo Ss. Two psychological treatments, stimulus-response compatibility (SRC) and stimulus discriminability (DISC), were introduced because they probably affect distinct information processing stages. Reduced stimulus DISC apparently impairs stimulus preprocessing and encoding operations, whereas low SRC impairs response selection and organization operations (Sternberg, 1969; Biederman & Kaplan, 1970). Ss were required to write down either the letter presented (high SRC) or the next successive letter in the alphabet (low SRC). In addition the letters, which were presented at an intensity level of approximately 83 db, were masked by a white noise of either 70 db (high DISC) or 80 db (low DISC). With errors of transcription as the dependent variable, alcohol (mean blood alcohol content = 100 mg percent) showed a significant interaction with SRC ($p < .01$) but not with DISC. These results suggested that at least one locus of the alcohol effect is the "selection and organization of responses." The additive effects of alcohol and DISC suggested that alcohol does not impair the operations involved in "stimulus preprocessing and encoding".

2. Sternberg's Memory Search Paradigm. A second experiment from our laboratory introduced alcohol in a memory search paradigm developed by Sternberg (1966; 1967). First, the S memorized a list of numbers. A series of probe digits was then presented visually, and S pressed one key if the probe digit was a member of the memorized set (M) and another key if it was not. When RT, the dependent variable, was plotted as a function of the size of M, more than 99% of the variance was accounted for by linear regression. Sternberg concluded that the slope of this regression function (about 38 msec) measured the time required to scan sequentially each item in M, while the intercept reflected the time required for preprocessing and encoding the probe digit as well as for response selection, organization, and execution. Sternberg (1967) superimposed a checkerboard grid (low DISC) over the probe digit on half the trials. In well-practiced Ss, this resulted in a systematic increase in the intercept, with no significant change in the slope (i.e., the treatments DISC and size of M were additive). Thus, he concluded, low DISC probably impairs the stimulus preprocessing and encoding stage but not the sequential scanning of M. A positive interaction between DISC and the alcohol treatment, therefore, would implicate "stimulus preprocessing and encoding" in alcohol effects, whereas a positive interaction between alcohol and the size of M would imply that alcohol impairs the scanning operation.

When an alcohol treatment was added to this experiment in our laboratory, alcohol produced no significant changes in either the intercept or the slope of the RT function and only minimally affected total RT. Furthermore, there were no significant interactions between alcohol and any other treatments. As in Sternberg's (1967) experiment, the effects of DISC and the size of M were additive, with DISC increasing only the intercept of the RT function, both in sober and intoxicated Ss. Thus, as in our earlier letter-recognition study, alcohol apparently did not impair "stimulus preprocessing and encoding". Moreover, the additive relationship between alcohol and the size of M indicated that speed of scanning of short-term memory was not altered by the drug.

Alcohol did significantly increase the error rate, however, especially the rate of incorrect responses to probe digits which belonged to the memorized list ($p < .01$). Performance was faster for these error trials than for correct trials, ($p < .01$, t test) the RT of incorrect responses to an M digit being 406 msec as compared with 466 msec for correct responses to an M digit or 438 msec for correct responses to other digits. Since the RT of 406 msec was much longer than simple visual RT (about 200 msec), we concluded that the intoxicated S was actually processing information during these error trials. However, the 60 msec increase in speed for incorrect M responses suggested that on these trials an information processing stage may have been skipped. A comparison of regression functions relating the RT of correct or incorrect responses to size of M showed that the 60 msec advantage on incorrect trials was due primarily to a reduction of the intercept. Moreover, there was no significant loss of effectiveness of the mask (low DISC) on incorrect trials. We concluded that the omitted stage was located on the response side of the system, probably the response selection and organization operations.

3. Stimulus-Response Uncertainty and Stimulus-Response Compatibility with Verbal Reaction Time. Huntley (1972), using an information processing approach to analyzing drug effects similar to that used here, found that alcohol interacts positively with a SRU treatment, defined by the number of different stimulus-response alternatives. Furthermore, Sternberg (1969) has reported that SRU and SRC interact positively, suggesting that both treatments influence a single stage of information processing, probably "response selection and organization". As a result, Sterngerg's experiment combining both of these treatments seemed to be an excellent paradigm within which to continue testing our hypothesis that alcohol affects the "response selection and organization" stages of information processing.

In this study, a digit was presented visually and Ss named the digit (high SRC) or the one next in ordinal progression (low SRC). The digit was either masked by a checkerboard grid (low DISC) or unmasked (high DISC). Finally, each block of 20 trials contained two (low SRU) or eight (high SRU) different numbers. We found that alcohol caused a significant increase in RT of approximately 60 msec, ($p < .001$) suggesting that the drug might specifically increase the time required for verbal operations. Furthermore, alcohol showed a significant ($p < .01$) positive interaction with both SRC and SRU (see Fig. 1). The three-way interaction between the alcohol, SRC, and SRU treatments was just short of the 0.05 significance level ($F_1, 17 = 3.90$, $p < .10$). Taken together, these results support our prior conclusions that alcohol impairs the selection and organization or responses. Moreover, the selection, organization, or execution of verbal responses may be particularly difficult for inebriated Ss. As in previous studies, there was no positive interaction between the alcohol and DISC treatments, again supporting the view that alcohol does not impair stimulus preprocessing and encoding operations. The pattern of additivity and interactions among established treatments was identical in both intoxicated and sober Ss to those found by Sternberg (1969).

In conclusion, the use of alcohol as a treatment in information processing paradigms has generally confirmed or strengthened the models used. No unusual patterns of interaction occurred between established treatments when alcohol was introduced. The patterns of additivity and interactions between alcohol and established treatments implicate output operations (e.g., "response selection and organization" and possibly the execution of verbal responses) rather than input operations (e.g., "stimulus preprocessing and encoding"), or other cognitive operations such as high-speed sequential scanning of a short-term buffer memory.

4. Current Research. Our current research with alcohol is an attempt to extend our finding that alcohol affects output operations. More specifically, since both information processing and memory research (see below) in our laboratory suggest that alcohol might impair the "selection and organization" of verbal responses, one possible site for the drug effect is the selection or retrieval of names from long-term memory storage.

According to the Sternberg procedure, in order to implicate a new treatment as affecting a particular stage of information processing, one must show that it interacts positively with a factor known to influence that particular stage. The rate at which words occur in the English language seems to exert a primary influence upon the "name retrieval" stage of information processing (Oldfield & Wingfield, 1965; Wingfield, 1968; Seymour, 1973). As a result, we are now determining whether or not alcohol interacts with this treatment.

There are several current models to explain the word frequency effect in terms of "name retrieval". Oldfield (1966) proposed that one's memory for object names is organized into ensembles, with the largest ensemble containing rare names, a somewhat smaller ensemble for more frequent names, and so on with the smallest ensemble composed of the most frequently used English words. Each ensemble is assumed to have an equal probability of occurrence. He then postulated that picture naming involves two successive operations: (1) an estimate of familiarity which requires a constant amount of time to select the appropriate ensemble, and (2) a dichotomous search of the chosen ensemble for the correct name. The amount of time required for the dichotomous search depends on the size of the ensemble which, in turn, depends upon word frequency.

Briggs and Swanson (1969), Wingfield (1968), and Fraisse (1969) have all provided experimental support for Oldfield's model. Nevertheless, despite this support, the model has a weakness due to its simplicity, since it predicts a word

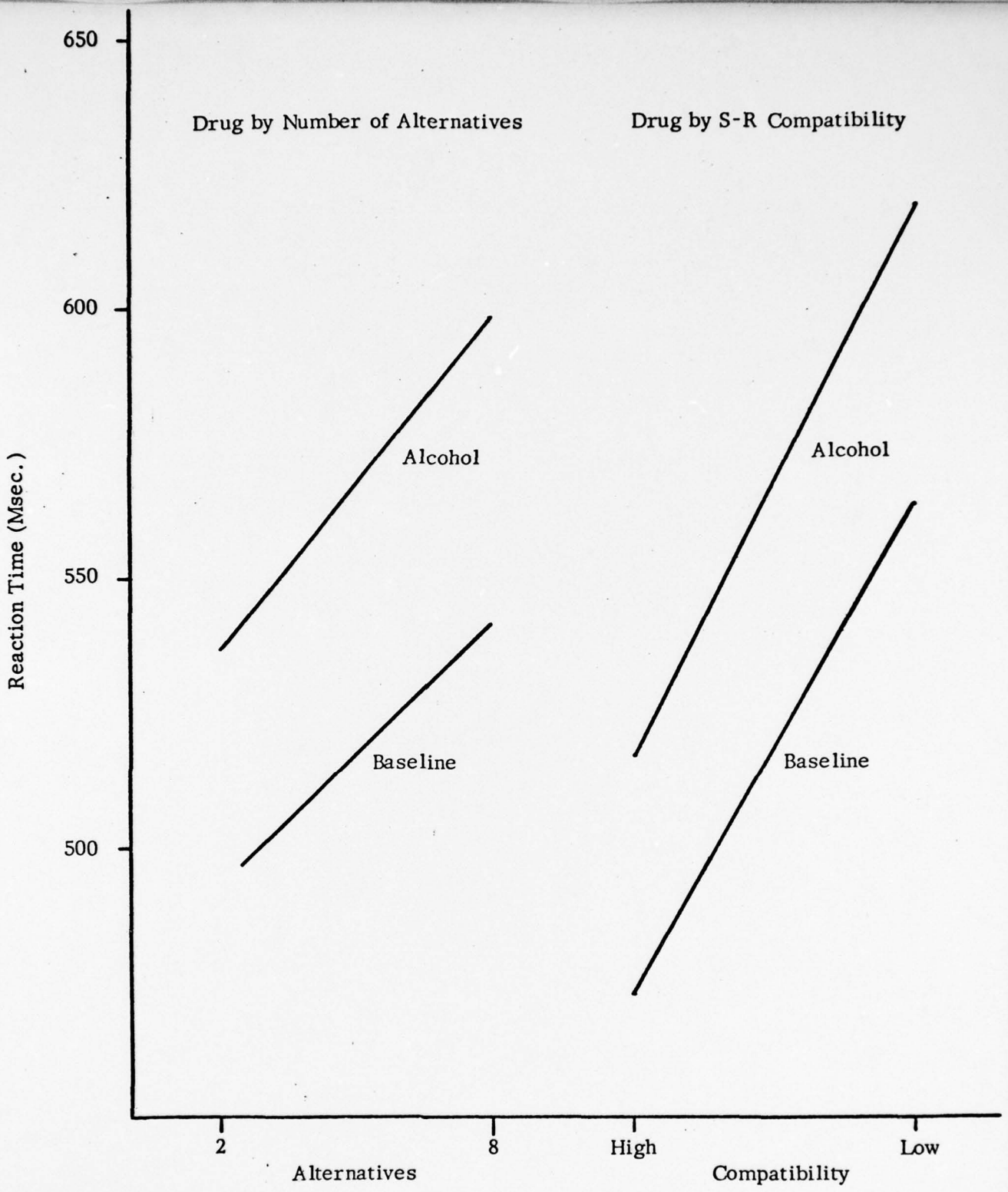

Fig. 1. Effects of alcohol, number of response alternatives, and S-R compatibility on verbal reaction time. The graph on the left illustrates the main effects of alcohol and number of alternatives and the interaction between that task variable and alcohol. The graph on the right shows the interaction between alcohol and stimulus compatibility task variable.

frequency effect only when a "name retrieval" (i.e., memory search) operation is necessary. It cannot handle the evidence, for example, that both RT (Pierce & Karlin, 1957) and visual detection thresholds (Howes & Solomon, 1951; Rosenzweig & Postman, 1958) for printed words are a function of the frequency of occurrence of the words, since "name retrieval" is not a necessary operation in these tasks.

Subsequently, Morton (1970) proposed a model that accounts for the word frequency effect on activities not involving a name retrieval operation. Seymour (1973) has expanded this model to account for the word frequency effects on tasks in which this operation is important, such as on RT to name objects.

Morton developed the Logogen model, as it is called, on the assumption that the same final unit operates to produce a specific verbal response regardless of the source of information that led to the response. The final unit is called a "logogen" and is defined by the sets of acoustic, visual, and semantic attributes which constitute its input. (In Fig. 2, only the visual and semantic input are represented.) Although a logogen is a word in terms of its output, in terms of its input it is a hypothetical counting device that triggers when an appropriate threshold is reached. Graphemic analysis of a word, for example, might produce the following attributes: "three-letter word", "initial letter /d/", and "final letter /g/", resulting in three counts for both "dog" and "dig". Since "dog" is the more frequent word, it would be nearer to its threshold and consequently a more probable response if required. Semantic attributes lower the number of sensory attributes necessary for the logogen to reach threshold. For example, the attributes "house pet" and "initial letter /d/" should readily trigger the "dog" logogen. Finally, the model postulates that no external decision process is required when a response is forced. Instead, thresholds are generally lowered until the logogen closest to its criterion fires.

Seymour (1973) argued that visual analysis of pictures probably requires a different system from that of words. As a result, his modification of the Logogen model included a pictorial analyzer and a pictorial logogen system. The pictorial logogen has little relevance in this discussion, since it mainly functions to produce visual output (e.g., a drawing).

The operations required in reading a word are essentially the same as Morton postulated and may be represented as follows:

graphemic analysis ----> operation of the word logogen ----> articulation planning ----> responding.

Naming an object, however, requires two additional operations in the following sequence:

pictorial analysis ----> operation of the pictorial logogen ----> semantic memory search ----> operation of the word logogen ----> articulation planning ----> responding.

Seymour accepted Fraisse's (1969) findings that there is no word frequency effect in reading words. As a result, he interpreted the familiarity effect in object naming tasks as being due to either or both of the additional operations, i.e., the pictorial logogen or semantic memory. This, however, will not explain the findings of Pierce and Karlin (1957) or our own pilot work which suggest that word frequency also affects the time needed to read words.

As has been shown, the logogen system in the model can explain the influence of word frequency on visual detection thresholds (VDTs). The same part of the model can also be used to account for the influence of word frequency on the time required to read words. A logogen for a low frequency word requires more attributes to reach threshold than does a logogen for a high frequency word. As the

"logogen system" requires more attributes, the "graphemic analyzer" may require more time to provide them. Since visual attributes may be processed in a parallel manner (Hawkins, 1969), the increase in RT essentially may be due to the addition of some attribute that requires more time to process. As more attributes are needed by the logogen system, the probability of having a slowly-processed attribute becomes greater. If such parallel processing occurs, however, the contribution of word frequency on the time required to read words may be slight, especially when compared to a system in which the time required to process each attribute is added sequentially.

Semantic attributes may be sequentially processed by the "logogen system for words." If so, then the time needed to trigger a specific logogen unit would be equivalent to the sum of the times necessary to process each attribute required by that unit. Since the logogen bias requires more attributes for less frequent items, considerably more time would be needed to respond to uncommon items than to common ones. As a result, a word frequency treatment could have a very large effect on any task requiring the transfer of information from "semantic memory" to the "logogen system for words."

As an alternative, the much slower RTs produced by naming uncommon pictured objects might be due to the word frequency treatment having a distinct effect on one of the additional operations required by Seymour for the object naming task (i.e., the "pictorial logogen" and the "semantic memory search"). For example, the "pictorial logogen" might have a bias like that of the "word logogen". Furthermore, the time required to search semantic memory for a particular item might be influenced by the number of times that search has been conducted in the past. In either case, this new word frequency effect would simply add on to any produced by the "logogen system for words".

Our current set of experiments using alcohol is designed to (a) determine which stage(s) (and which model) best account for the word frequency effect, and (b) whether alcohol interacts with word frequency and thus also influences the implicated stage(s).

Moskowitz and Roth (1971) reported that both alcohol and low frequency names of pictured objects generally slow verbal RT. They also found that alcohol interacts positively with word frequency. We are conducting a modification of the Moskowitz-Roth task, using both outline drawings and printed words. The Ss are told to read the words and name the pictures as quickly and as accurately as possible. Each S is run four days: the first session being practice; the second and fourth being baselines; and the third session being under alcohol (1.056 g/kg). On each session Ss view a different set of 110 stimuli. Words and pictures occur equally often in the set in counterbalanced blocks. Furthermore, each stimulus category is represented equally by each of the five word frequency groupings of Moskowitz and Roth as determined from Thorndike and Lorge (1944). All stimuli are presented twice in each session. We are also measuring the distribution of the outline drawings in each word frequency grouping along an "uncertainty" dimension used by Lachman (1973). This measure, defined by the number of different responses given to a particular stimulus on its first presentation by all Ss may be a better predictor of RT than word frequency.

Seven of 24 total Ss have been run. The results from these Ss indicate that both alcohol and word frequency generally slow RT. The latter finding, confirming Pierce and Karlin (1957), suggests that Oldfield's model is not adequate to handle all of the data, and thus must be rejected in favor of the Morton-Seymour model.

The primary purpose of this experiment is to determine the effects of alcohol on those operations found to be influenced by word frequency. Trends cannot be analyzed at this time since the data from all Ss will be needed to determine the

"uncertainty" dimension. However, the possible interpretations are outlined below.

If alcohol increases RT to uncommon stimuli equally in both picture naming and reading, then it probably affects just the bias of the "word logogen system." Since Morton (1970) postulates that the bias for particular logogens is lowered by repetition, then an alcohol effect on this system would probably also alter the effect of repetition. On the other hand, if alcohol increases the effect of word frequency on naming more than on reading, then the drug probably also affects some operation unique to naming. The choice between the two most probable stages--the "pictorial logogen" or "semantic memory search"--would depend upon whether or not alcohol increases the influence that high uncertainty has on RT, respectively. Since the "pictorial logogen" probably exerts a bias similar to its word equivalent, an alcohol impairment of this system might alter the effect of repetition on word frequency in the naming task.

B. Barbiturates.

Studies of human performance effects of barbiturates are not extensive, but most investigators find that practically all of their performance measures are impaired. These agents slow RT (Goldstein et al., 1960), impair time estimations of short intervals (Rutschmann & Rubinstein, 1966; Goldstone & Kirkham, 1968), disrupt memory when little stress is required in the task (Evans & Davis, 1969), and may impair any task requiring an intense associative or cognitive effort (Mirsky & Kornetsky, 1964). Together, these studies do not provide any systematic pattern of results. Therefore, we have begun a systematic information processing analysis of one barbiturate, secobarbital sodium. In addition, we are interested in whether or not secobarbital influences the same outputting functions that are impaired by ethyl alcohol.

In two experiments (Rundell et al, in preparation) we have introduced secobarbital in the Sternberg memory search paradigm and the Sternberg stimulus compatibility-stimulus discriminability paradigm--both of which were used with alcohol. We have just completed testing the effects of secobarbital on the Posner and Mitchell (1967) RT method for measuring various levels of stimulus encoding. These results are considered below.

1. Sternberg's Memory Search Paradigm. The RT experiment reported above was repeated with secobarbital sodium (200 mg/70 kg body weight) or d-amphetamine (15 mg) replacing the alcohol treatment. In addition, a within Ss design was used in which Ss were compared with their own baselines. The significant effects for memory set (M) size and stimulus DISC found in the alcohol study were reconfirmed ($p < .001$, F tests). In addition, response type (R) had a significant ($p < .05$) overall effect on RT in that "yes" responses were slower than "no" responses.

The effects of M and DISC on RT were additive, suggesting that they influenced separate information processing stages. When the regression of M size on RT was calculated, neither secobarbital nor amphetamine produced a significant change in slope from the baseline. Thus, like alcohol, neither drug affected the serial memory interrogation operation. D-amphetamine did not significantly affect the intercept, although it produced a small, nonsignificant ($F_{1,30} = 1.23$) decrease in overall RT. Furthermore, it did not interact with any treatment used.

Secobarbital produced a much larger increase in overall RT (58 msec) than had been produced in the same experiment with alcohol (21 msec). In addition, the drug interacted positively with two different treatments used in the design, DISC and R. Figure 3 shows that the masked condition resulted in a 20 msec greater increase in RT under secobarbital than under baseline conditions ($p < .05$,

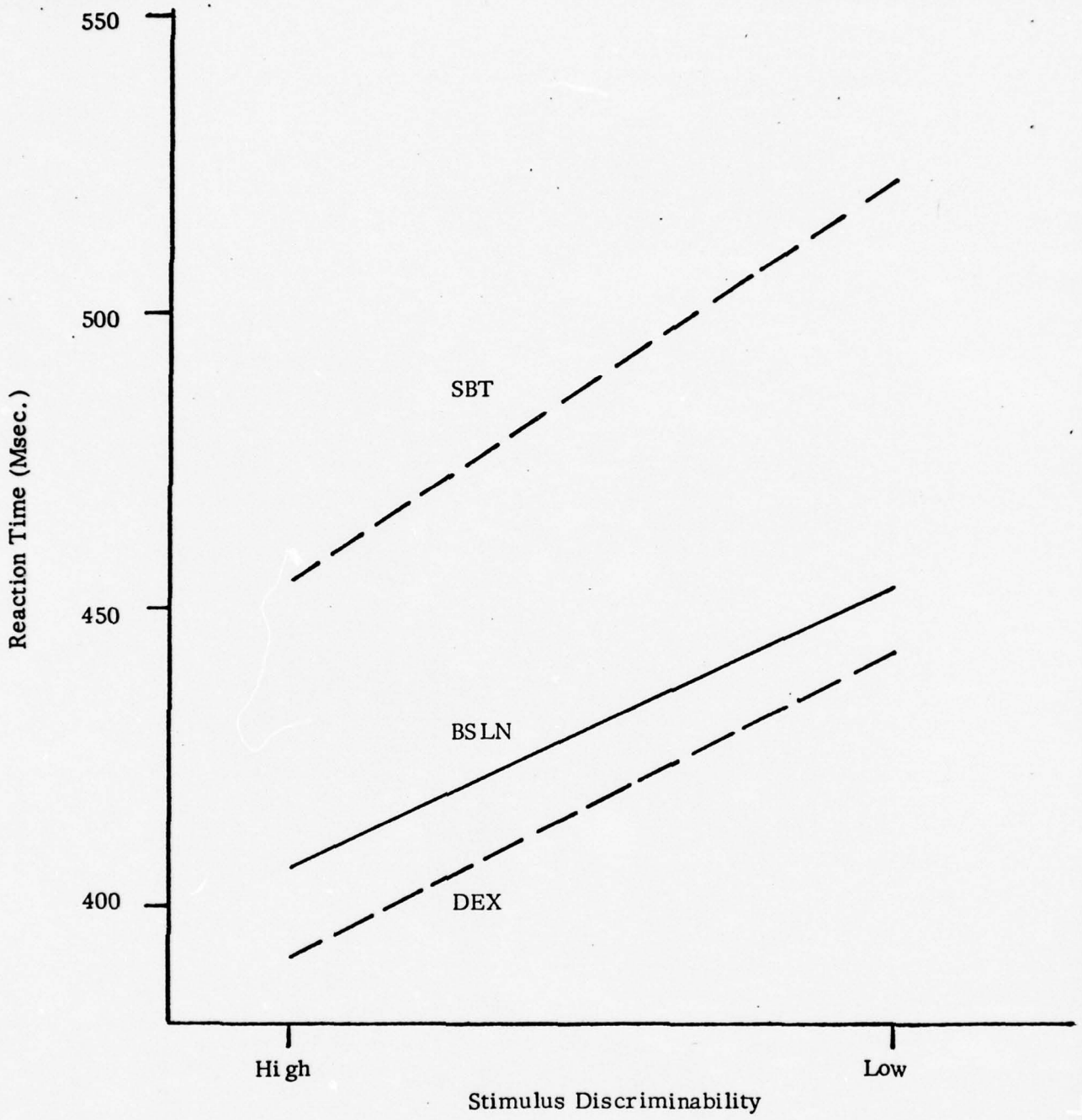

Fig. 3. Interaction between secobarbital and stimulus discriminability on the Sternberg memory search task. This graph illustrates that secobarbital (SBT) showed a greater effect on reaction time due to low stimulus discriminability than either d-amphetamine (DEX) or the baseline (BSLN).

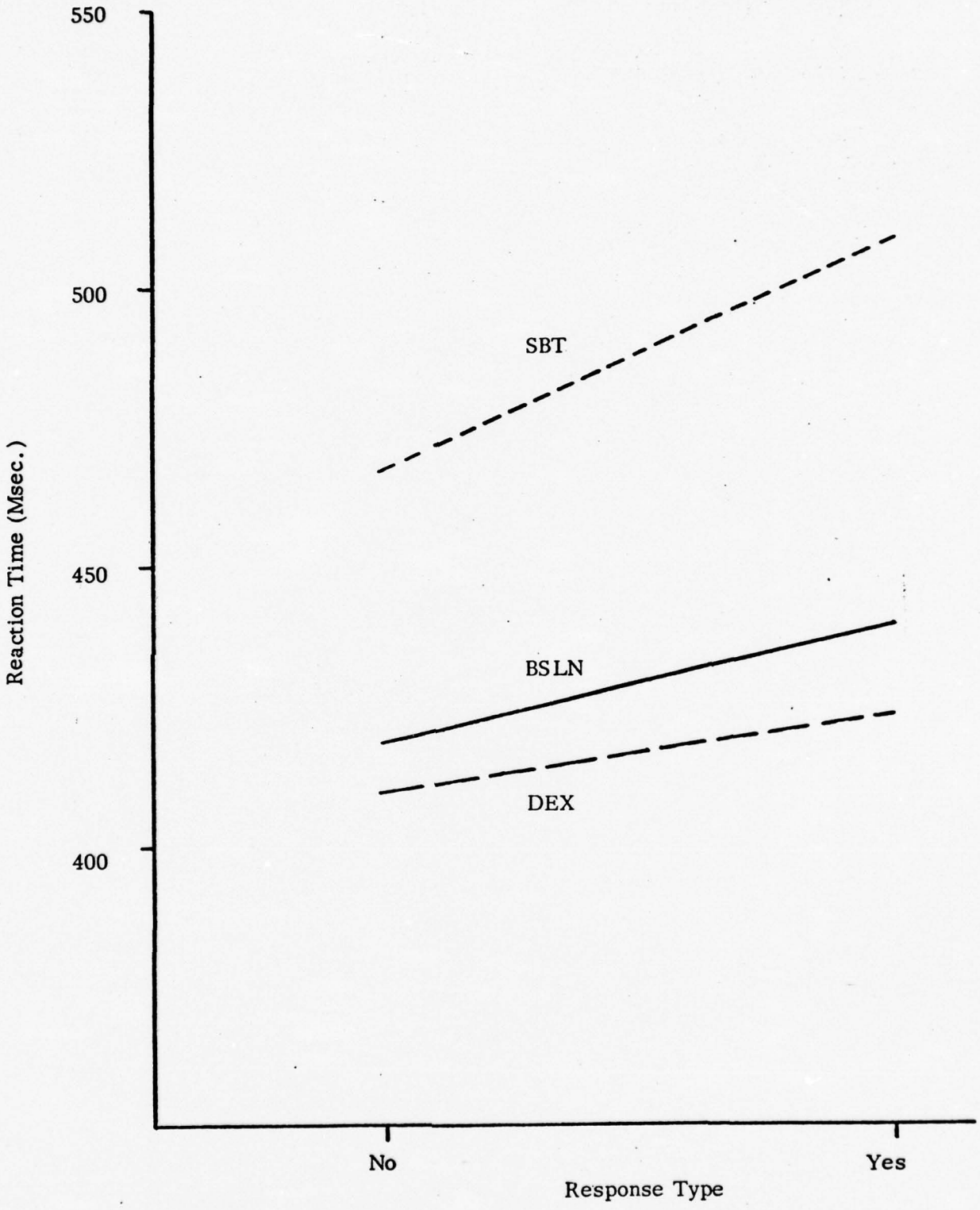

Fig. 4. Interaction between secobarbital and response type. This graph illustrates that yes responses slowed reaction time more in the secobarbital (SBT) condition than in either the baseline (BSLN) or d-amphetamine (DEX) conditions.

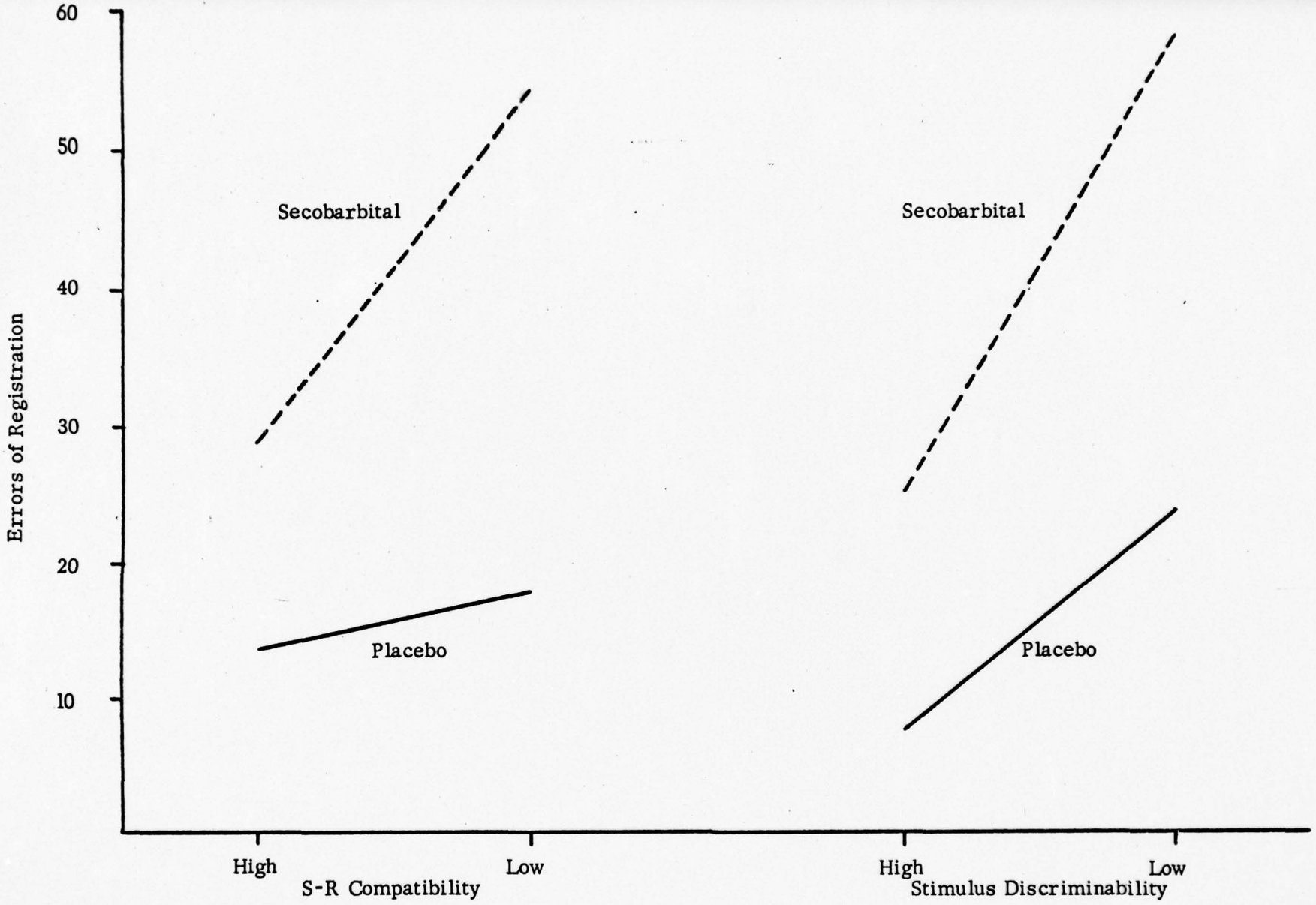

Fig. 5. Interactions of secobarbital with S-R compatibility and stimulus discriminability.

F test). In addition, Figure 4 shows that "yes" responses were 20 msec slower than "no" responses under secobarbital as compared with baseline ($p < .05$).

In accordance with Sternberg's method of analysis, these results suggest first that secobarbital affects a stage of information processing which is also sensitive to stimulus discriminability, (i.e., stimulus preprocessing and encoding). This is quite a different impairment from that produced by alcohol, vis., an alteration of response selection and organization operations.

The secobarbital by R interaction further suggests that a later stage of information processing is also slowed by secobarbital. According to Sternberg (1969) this stage may be "response selection and organization". If so, then alcohol and secobarbital influence this stage in common. When alcohol was used as a treatment in this paradigm, however, we did not find a significant overall difference in RT between "yes" and "no" responses. As a result, we also failed to find an alcohol by R interaction in the alcohol study. To eliminate a practice session, we continued to run most of the alcohol Ss in the secobarbital experiment. The overall significance of R and the significant slowing of "yes" responses under secobarbital may be due to well-practiced Ss paying greater attention to response probabilities in this study.

2. Stimulus Compatibility and Stimulus Discriminability. If both "stimulus preprocessing and encoding" and "response selection and organization" are affected by secobarbital, then the drug should interact with both treatments in our first core experiment (see above). In this study tape recorded letters were presented under a high (low DISC) or low (high DISC) background noise, and the Ss responded by writing down the letter presented (high SRC) or the next letter in the alphabet (low SRC). Sixteen secobarbital Ss, like previous alcohol Ss, showed an overall increase in errors of transcription as compared with 16 placebos ($p < .001$, F test). Low DISC and low SRC both produced overall increases in errors ($p < .001$). Furthermore, DISC and SRC proved to be additive, reconfirming previous findings, that they influence separate information processing stages.

Figure 5 shows the effect of secobarbital on the two treatments. Both interactions are significant ($p < .05$) with an analysis of both the raw error scores and with a square root transform. Secobarbital increased the discriminability slope and the SRC slope by 16 and 21 errors, respectively, over the placebo. These results confirm those of the previous experiment in suggesting that both "stimulus preprocessing" and "response selection" are affected by secobarbital.

3. Matching at Various Levels of Processing. Posner and Mitchell (1967) have developed a RT method for measuring various levels of stimulus encoding. Ss are presented with pairs of letters and asked to respond according to whether the letters are the "same" or "different". By varying the criterion for sameness, the required degree of encoding may be controlled. A "same" response may be based on physical identity (e.g., A, A), at a higher level on name identity (e.g., A, a), and at a still higher level of rule identity (e.g., both vowels). This procedure requires that, at any particular level of processing, no lower level response be possible (e.g., when a name-match is required, a physical identity match will never be presented).

The Sternberg experiment does not enable us to determine whether or not a physical or a name code is used by the Ss. We were interested in determining at what level "stimulus preprocessing and encoding" might be retarded. If the drug impaired operations at the level of physically encoding the stimulus, then one would expect all levels of processing in the Posner-Mitchell paradigm to be slowed under secobarbital. On the other hand, if encoding were influenced only at a level of verbal processing, we would expect physical matching to be unimpaired by the drug.

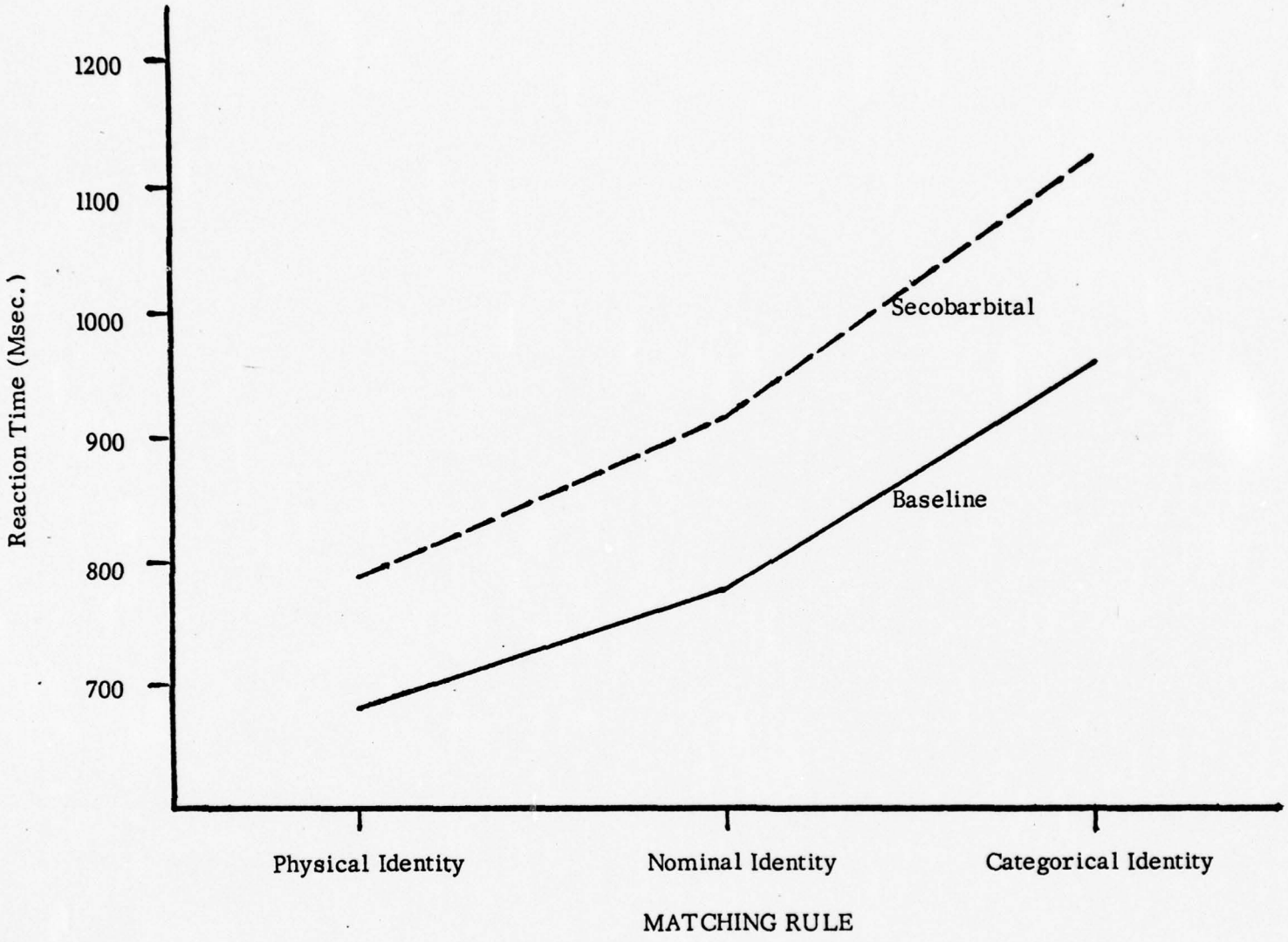

Fig. 6. Additivity of secobarbital and type of matching on the Posner-Mitchell task.

Eleven males and 11 females were given baseline and secobarbital treatments using this task. Secobarbital, level of matching, and response type (i.e., "same" vs "different") all significantly ($p < .001$) increased RT. Furthermore, all of these treatments produced additive effects on RT. Although secobarbital produced an overall increase in RT of about 140 msec, it showed only a slight, nonsignificant tendency to increase RT more at higher levels of processing ($F_{2,36} = 1.85$). Figure 6 compares secobarbital and baseline conditions across the three levels of matching. These results suggest that the drug slows down the encoding process at its most rudimentary level--that of physically encoding the stimulus.

One additional finding is that the drug affected males more than females. Secobarbital slowed overall RT by almost 200 msec in males as compared to only 69 msec in females. Furthermore, the female reaction did not appear to be a function of their menstrual cycle. Two possible explanations might account for this effect. First, the secobarbital dosage was given according to body weight, so males generally received more of the drug than females. Secondly, females have more fatty tissue than males and thus may absorb the drug differently.

In summary, the effects of secobarbital on information processing are apparently more complex than those of alcohol. Our data imply that the barbiturate not only impairs cognitive processes associated with response selection but also very early processes on the input side, operations associated with preprocessing and encoding of the stimulus.

C. Experiments Focusing on Memorial Processes.

Over the past decade, experiments in a variety of laboratories have indicated that memory consists of two or more functionally discrete stages having differing characteristics (Glanzer 1972; Atkinson & Shiffrin, 1968; Murdock, 1967; Waugh & Norman, 1965, Sperling, 1963, 1967; Broadbent, 1958, 1969). Perhaps the best known and most comprehensive model is that of Broadbent (1969) which postulates three separate memory stores: (1) a perceptual time-dependent buffer store from which items are lost by "decay", (2) a limited capacity short-term store (STS) from which items are displaced by the entry of new items, and (3) an unlimited capacity long-term store (LTS). With respect to memory, sections A and B (above) apply most directly to perceptual memory and STS; whereas, the free recall experiments are most directly applicable to the study of STS and LTS. Thus the separate experiments discussed above and in this section provide differing areas of focus while encompassing a wide range of memory functions.

In line with the general experimental strategy discussed in the preceding sections, we have selected a viable model--Glanzer's (1972) adaptation of Broadbent's model--and conducted drug experiments within the framework of that model. As illustrated by Figure 7, the model arrays STS and LTS serially such that items can be entered in LTS only by way of STS. (The transfer of an item from STS to LTS does not, however, remove the item from STS.) The capacity of STS is approximately 2 ± 1 items, and items are removed from STS only through "displacement" by a new entry. The removal of items from STS operates as a probabilistic approximation of the "first in-first out" principle; and the probability of any item remaining in STS can be expressed as 0.5^N, where N is the number of subsequent items entered in STS. Access to STS is relatively direct as compared to LTS access. Items entered in LTS may be lost by some unspecified process (e.g., decay) or they may simply become inaccessible to the retrieval mechanism.

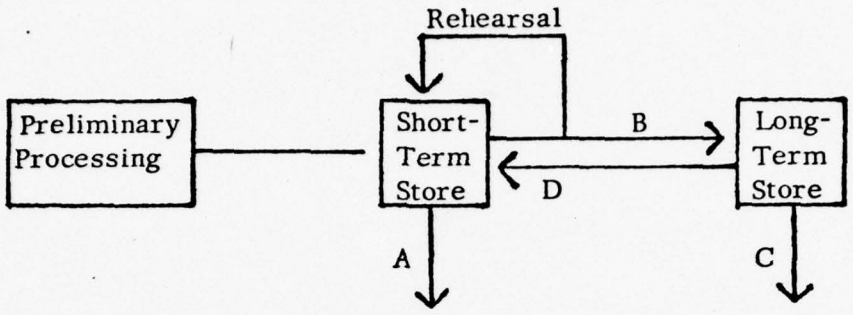

Fig. 7. Glanzer's (1972) model of information processing in the free recall paradigm. Preliminary Processing may include stimulus preprocessing and encoding and a "very short-term" memory store (see, Sperling, 1960). The arrow labeled "A" represents two processes: (1) the loss of items from STS by displacement; and (2) STS retrieval mechanisms. The recursive arrow on STS represents rehearsal operations which reenter items in STS and which can occupy some of the channel capacity used for transferring items to LTS. Arrow "B" represents the STS-to-LTS transfer operation and does not imply a loss of items from STS. Arrow "C" indicates the possible loss of items from LTS. Arrow "D" represents retrieval from LTS and indicates that items so retrieved are entered in STS (although the process may not involve direct entry in STS).

In the paradigm used in our laboratory, S is visually presented a series of English words. At the end of the series (list) he is asked to recall as many of the words as he can, without regard to order or presentation (free recall). The task is then repeated several times with different lists or with the same list with the words shuffled. When the empirically determined probability of recall is plotted as a function of the serial position of the words in the list, the typical U-shaped curve is found. It has been shown (Murdock, 1962; Glanzer & Cunitz, 1966; Postman & Phillips, 1965; Glanzer & Schwartz, 1971) that recall of items in all but the last few positions of the list is affected by the classical rote-learning variables: (1) rate of presentation, (2) word frequency, (3) list length, and (4) mnemonic or associative structure. The only task variable, however, which has been found to affect recall of the final few items of the list is the input of additional information prior to recall. Moreover, this variable does not affect recall of earlier items in the list. Thus, the analysis of serial position recall functions enables the experimenter to differentiate treatment effects on the short- and long-term storage mechanisms. Any variable which affects all but the last few items in the list is assumed to affect LTS, and any variable which affects recall of the final few items presumably affects STS.

As mentioned above, previous research has identified a number of task variables which differentially affect the mechanisms related to STS and LTS. It has been shown, for example, (Glanzer & Meinzer, 1967) that when the interval between presentation of items A and B is filled with a forced "rehearsal" (multiple verbal repetitions) of item A, the probability of item A being in STS is unaffected; but the probability that item A will be transferred to LTS is substantially reduced. This reduction in the transfer of items to LTS may be interpreted as the result of the partial occupation of the available channel capacity by the forced rehearsal task. Thus, forced inter-item rehearsal can be employed as a task variable to test the hypothesis that a drug impairs memory by reducing the channel capacity available for transferring words from STS to LTS.

A second variable which previous research has shown to affect recall from LTS is list length. Through what may be a retroactive inhibition mechanism, the larger the number of items registered in LTS, the lower the probability of recall of any given item (Postman & Phillips, 1965). Some question remains as to the precise mechanism involved; but one strong possibility is that when a large number of items are entered in LTS in close temporal contiguity, they cannot be efficiently organized or indexed, and thus cannot be readily retrieved.

A third task variable, mnemonic or associative structure, has been shown to affect only LTS (Glanzer & Schwartz, 1971; Murdock, 1963; Phillips, et al., 1967). This variable is generally believed to affect recall by varying the difficulty of organizing items in memory and, thus, the difficulty of retrieval.

The only task variable which has, so far, been shown to affect STS is the interpolation of items prior to recall of the list (Glanzer, et al., 1969). The important factor in this task is the number of new items entered in STS. Passage of time, the information load of interpolated items, and the similarity of the interpolated items and the memory list items have no effect on clearing STS (Glanzer, 1972). Moreover, the interpolation of input between list presentation and recall has no effect on LTS. Thus, comparison of recall with and without an interpolated task enables the experimenter to isolate the amount of information actually held in STS. The effect of an interpolated task on the serial position recall function is to lower the right-hand portion of the curve to the approximate level of the middle of the list.

1. Effects of Alcohol on STS and LTS. As discussed above, previous research has established a number of factors which affect various aspects of the memorial processes. The study of drug effects within the model, thus, is primarily a search for patterns of additivity and non-additivity (as evidenced by statistical interaction) among the task variables and the drug. A basic assumption is that when two factors affect separate stages, their effects will be additive. Generally, it may be assumed that factors affecting the same stage will have non-additive effects.

Experiment 1. An initial free recall experiment, reported last year, employed the task variables, list length, forced inter-item rehearsal, and a 15-second delay interval interpolated between the presentation of the list and recall. The delay interval was either (1) unfilled (S sat quietly), or (2) filled by a counting backwards task. S received either a placebo or sufficient 95% ethanol, mixed with orange juice, to produce a mean blood alcohol concentration BAC or 100 mg percent.

For both alcohol and placebo conditions the effects of the task variables replicated previous research and, thus, added support and generality to the earlier findings. Alcohol substantially impaired recall ($p < .001$, F test) and did not interact with serial position ($F = 0.2$). This latter finding indicated that alcohol had impaired recall from both STS and LTS. The probability of items being held in STS was calculated to confirm the STS effect and alcohol was found to significantly reduce this probability ($p < .05$, t-test). There was also a tendency ($p < .10$) for alcohol to interact with the task variable, list length.

These results were interpreted as indicating that alcohol impaired both STS and LTS, but did not affect the mechanism transferring items from STS to LTS since the interaction of alcohol and forced inter-item rehearsal was not significant ($F_{1,30} = 1.51$). The trend toward an interaction between alcohol and list length suggested that items entered in LTS may not have been well organized or indexed, thus increasing the difficulty of retrieval. Analysis of errors of commission (i.e., incorrect responses made during the recall period) supported the notion that alcohol impaired the retrievability of items in LTS.

Previous studies (see, Jones, 1972) had indicated that alcohol has little effect on STS. We were, therefore, somewhat surprised by our contrary findings and felt that the unfilled delay interval could have led to an incorrect interpretation of our results. The 15-second unfilled delay interval might have been used by S to (1) transfer the final few list items, initially held in STS, to LTS and (2) then to rehearse words from the earlier portions of the list (i.e., words already in LTS). Such a procedure would displace the last few words in the list from STS, but would have little effect on the serial position function. In such a case alcohol could apparently impair STS while, in fact, simply be impairing recall from LTS. For these reasons a second free recall experiment was conducted.

Experiment 2. This experiment represents a partial replication of Experiment 1, but with one significant change: recall was initiated immediately following list presentation. As in Experiment 1, the task variables were list length (12 or 18 words) and forced inter-item rehearsal (1 or 5 verbal repetitions of each word). Eighteen Ss were run in a design with repeated measures on all variables except order of drug/placebo treatments. Order of drug/placebo treatments was counterbalanced, and sessions were separated by at least one week. The alcohol dose was varied according to body weight to achieve a mean BAC of approximately 100 mg percent.

For analysis, recall was averaged over blocks of the first, middle, and last four serial positions. The results of this experiment essentially duplicated those of Experiment 1. Alcohol and list length produced significant main effects ($p < .001$, F tests), as did forced inter-item rehearsal ($p < .05$). Forced inter-item rehearsal and list length interacted ($p < .001$) with serial position (see Fig. 8) confirming those findings in Experiment 1 and in previous research elsewhere, and supporting the notion that these variables affect LTS but not STS. As found previously, forced inter-item rehearsal and list length did not interact with each other, strengthening the notion that these variables affect separate stages. The alcohol x list length interaction (a trend in Experiment 1) was significant at the 0.05 level (see Fig. 9). As in Experiment 1, alcohol did not interact with forced inter-item rehearsal ($F = 0.3$) nor with serial position ($F = 0.7$). Alcohol significantly reduced recall at the final two list positions ($p < .005$, t-test) confirming that the drug affects STS. Analysis of errors of commission is currently in progress.

In summary, both free recall experiments showed STS and LTS deficits in intoxicated subjects. Since our model postulates that items are transferred from STS to LTS, an impairment of STS might be expected to reduce the number of items transferred to LTS. However, the interaction of alcohol and list length, along with the increase in prior list intrusions (Experiment 1) indicate a second and more direct impairment of LTS by alcohol. This latter effect may relate to an impairment of organization and retrieval of items newly entered in LTS.

Experiment 3. Jenkins and his colleagues (see, for example, Jenkins & Russell, 1952; Jenkins et al., 1958; Hyde & Jenkins, 1969; Johnson & Johnson, 1971) have hypothesized that associative linkages can be used to improve the organizational aspects of long-term memory, thereby facilitating retrieval operations. Within the free recall paradigm support for this hypothesis was provided by Glanzer and Schwartz (1971) who demonstrated that varying associative structure produced effects on LTS but not on STS. Since Experiments 1 and 2 indicated that one effect of alcohol on LTS might be the impairment of organization and retrieval, a third experiment was conducted to test the organizational impairment hypothesis. The major independent variables in this study were drug (alcohol/placebo) and strength of associative linkages. It was hypothesized that these variables would interact such that the combination of alcohol and low associative strength would have non-additive effects.

Experiment 3 was modeled along the lines of the classical free recall learning (free learning) paradigm: A list of words is presented and followed immediately by free recall. The list is then shuffled and presented a second time, etc. In the present experiment, three lists of 25 words were presented six successive times each, with free recall following each presentation. Each list was composed of two filler words followed by 10 word-pairs of known association value and three filler words at the end of the list. Each of the word-pairs (e.g., salt, pepper; black, white) were separated and the conglomerate of 20 words was then randomly shuffled with the constraint that no word-pair appear in either forward or reverse sequence. List 1 contained 10 word-pairs with a mean association value of 0.69 according to the Jenkins et al. (1958) norms. The mean association value of the word-pairs in lists 2 and 3 were 0.41 and 0.12, respectively. Eighteen Ss received the standard dose of alcohol, producing a mean BAC of approximately 100 mg percent; they were tested at that level. A second group of 18 Ss received a placebo drink consisting of 10-15 ml ethanol floated on top of orange juice. Both groups received six successive trials on each of the three lists, order of list being counterbalanced.

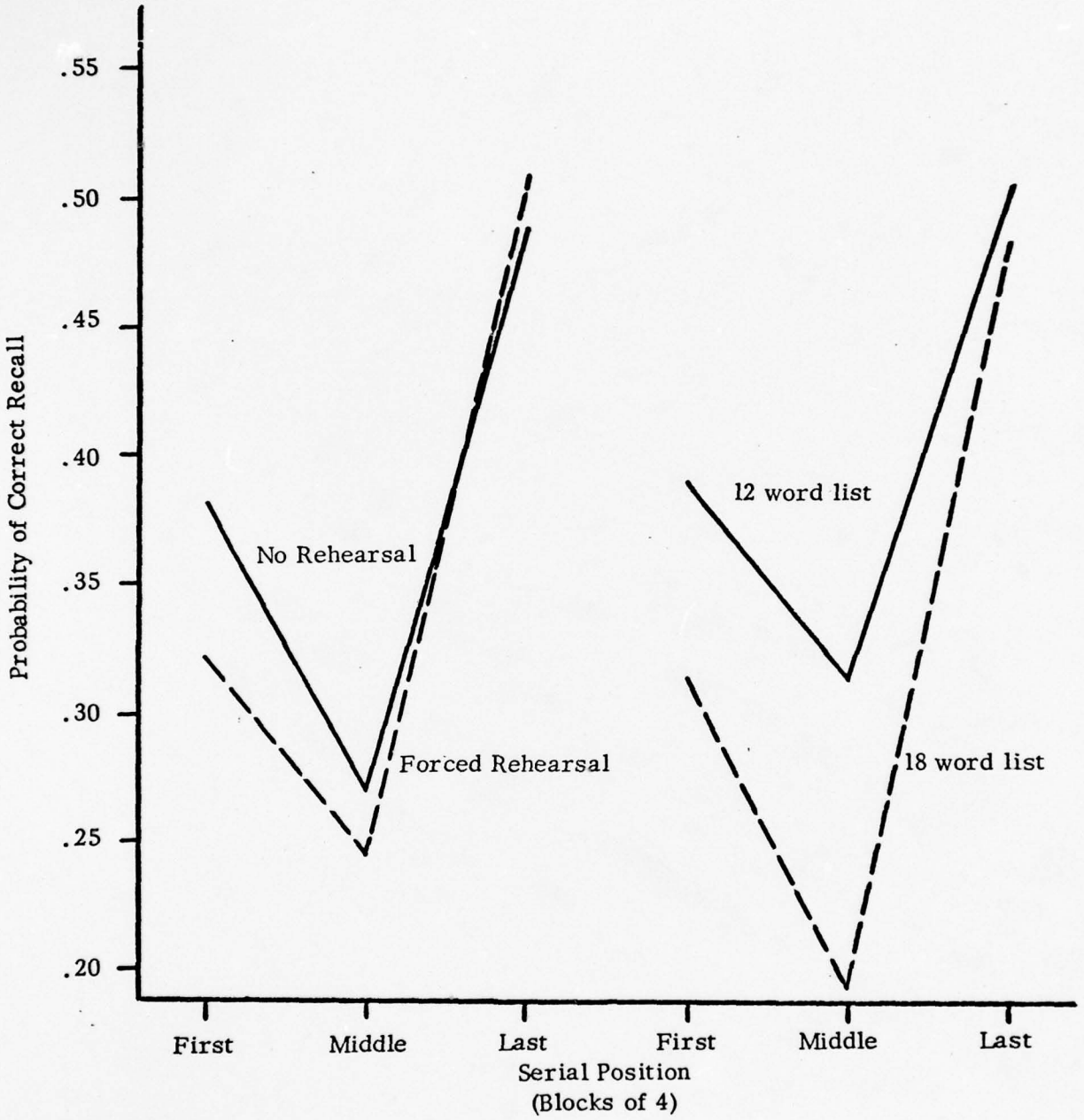

Fig. 8. Interactions of list length and forced inter-item rehearsal with serial position. The graph on the right illustrates the list length by serial position interaction ($p < .001$) and shows the effect of list length on LTS (all but the final few positions in the list). The graph on the left shows the similar interaction between forced inter-item rehearsal and serial position ($p < .001$).

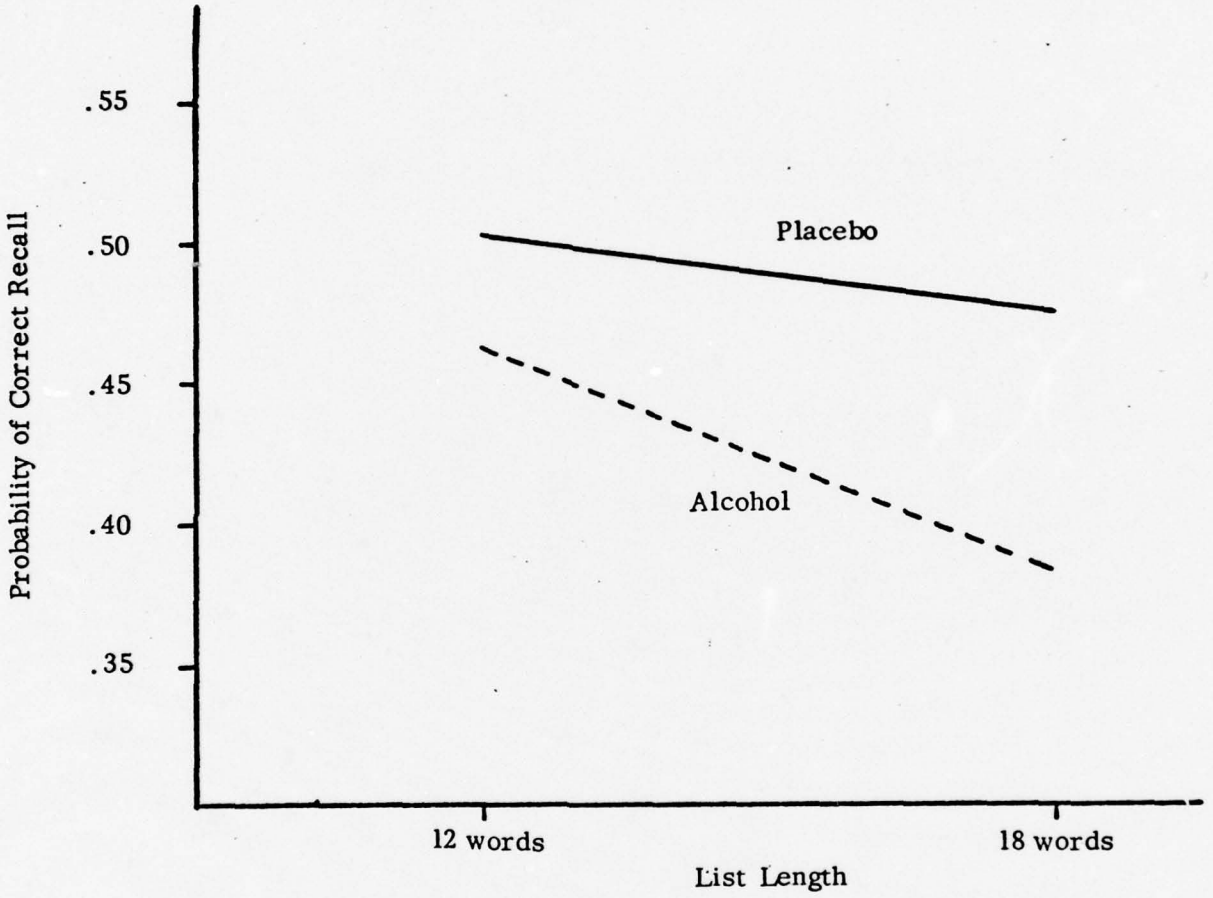

Fig. 9. Interaction of alcohol and list length ($p < .05$). Alcohol impaired recall of both the 12 word lists ($p < .05$) and the 18 word lists ($p < .001$).

Analysis of the data is not complete, but some of the results can be reported at this time. In the free learning paradigm, the initial presentation of a list does not differ procedurally from the free recall paradigm. Therefore, STS and LTS can be isolated by analysis of the serial position curves.

Analysis of the first presentation of each list. Based on the results of Experiment 1 and a large number of independent studies in other laboratories (Glanzer, personal communication) the last three words in each list were presumed to have been recalled from STS, whereas words in earlier list positions were presumed to have been recalled from LTS. Alcohol significantly ($p < .05$, t-test) reduced the probability of recall of words in the final three serial positions from a mean of 0.75 ($s = .18$) to 0.63 ($s = .19$), thus confirming the effect found in Experiments 1 and 2 and strengthening the hypothesis that alcohol impairs STS.

Figure 10 illustrates the effects of alcohol and association value on recall from LTS. It can be seen that alcohol substantially impaired recall from LTS at all levels of association value ($p < .0005$, t-test). The effect of association value is less clear. In the placebo group both high and intermediate levels of association value (lists 1 and 2) improved recall as compared with the low level of association value (list 3) ($p < .005$, t-tests); but the high and intermediate levels were not significantly different ($t = 0.07$. Within the alcohol group, recall of high association value words was significantly better than intermediate or low value words ($p < .025$, t-tests); but recall of the intermediate words was not significantly different than recall of the low association value words ($t = 0.8$). Thus, on the initial presentation of the lists, there was an overall effect of alcohol and of association value; and the two variables interacted significantly, indicating that they affect the same operation or stage. As can be seen in Figure 10, increasing the association value produced a much larger overall effect in the placebo group than in the alcohol group. These results suggest that alcohol impairs the ability to utilize associative linkages in organizing memory such that increasing the strength of the linkages has little effect on recall in intoxicated Ss.

Analysis of recall across repeated presentations of the lists. For this portion of the analysis, the five filler words (two at the beginning and three at the end of the lists) were discarded, and analysis of variance was performed on the proportion of the paired associates correctly recalled. As mentioned above, recall of these words is presumably from LTS. Main effects for drug, association value, and trials (six presentations of each list) were each significant at the 0.001 level. A strong interaction ($p < .001$) between drug and trials (see Fig. 11) indicates that recall improved more rapidly in the placebo group than in the alcohol group. Figure 12 shows the three-way interaction between association value, drug, and trials ($p < .01$) indicating that recall is substantially impaired by the combination of alcohol and low association value. In the alcohol group, the rate of learning from trial 1 to trial 6 is a function of association value. For the placebo Ss, association value affected the initial recall trial, but subsequently the rate of learning was about the same for each list. Thus, although alcohol reduced the effect of association value when the lists were presented the first time, the intoxicated Ss were able to make use of the associative linkages when given repeated exposures to the lists. Perhaps alcohol interferes with the development of a scheme for organizing items in memory as well as with the execution of the scheme. Sober Ss, on the other hand, may be able to respond quickly to associative linkages even though the linkages are weak.

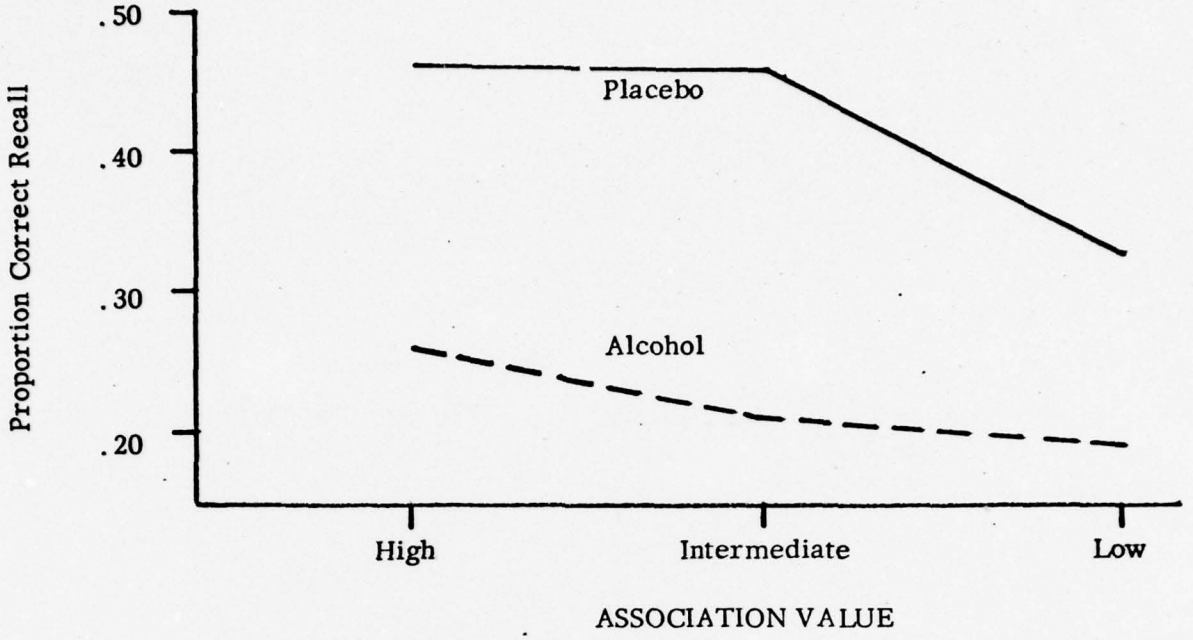

Fig. 10. Interaction of drug and association value for the first presentation of the lists (Trial 1).

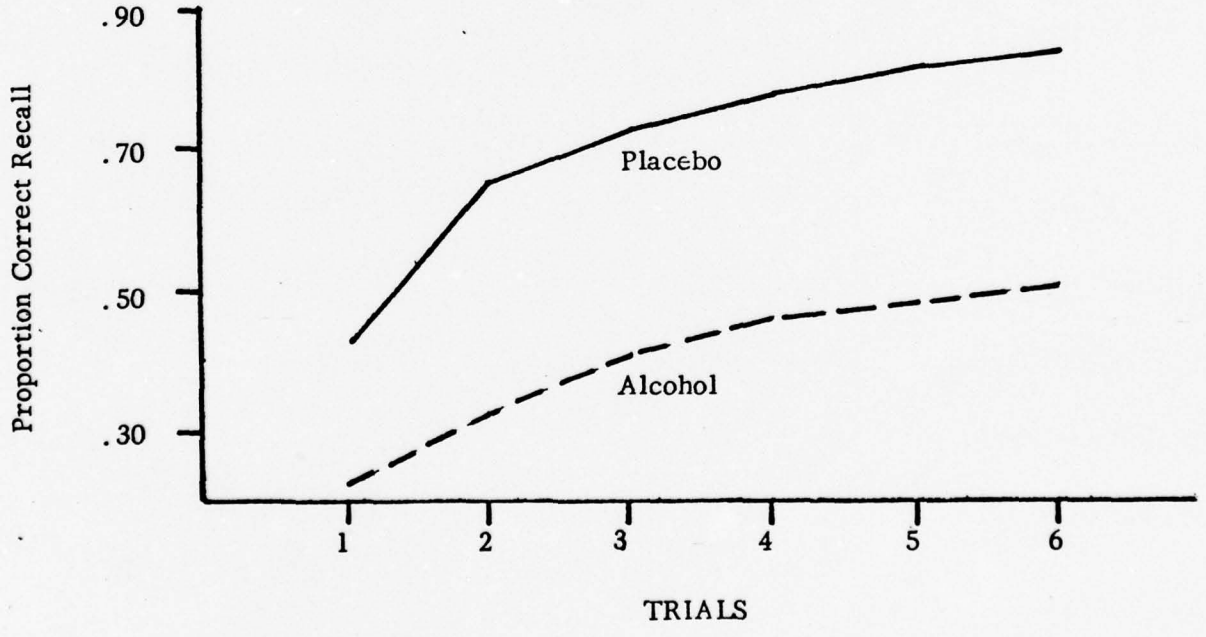

Fig. 11. Interaction of drug and trials on recall of associated word-pairs. The simple main effects of drug are significant at all levels of trials ($p < .001$).

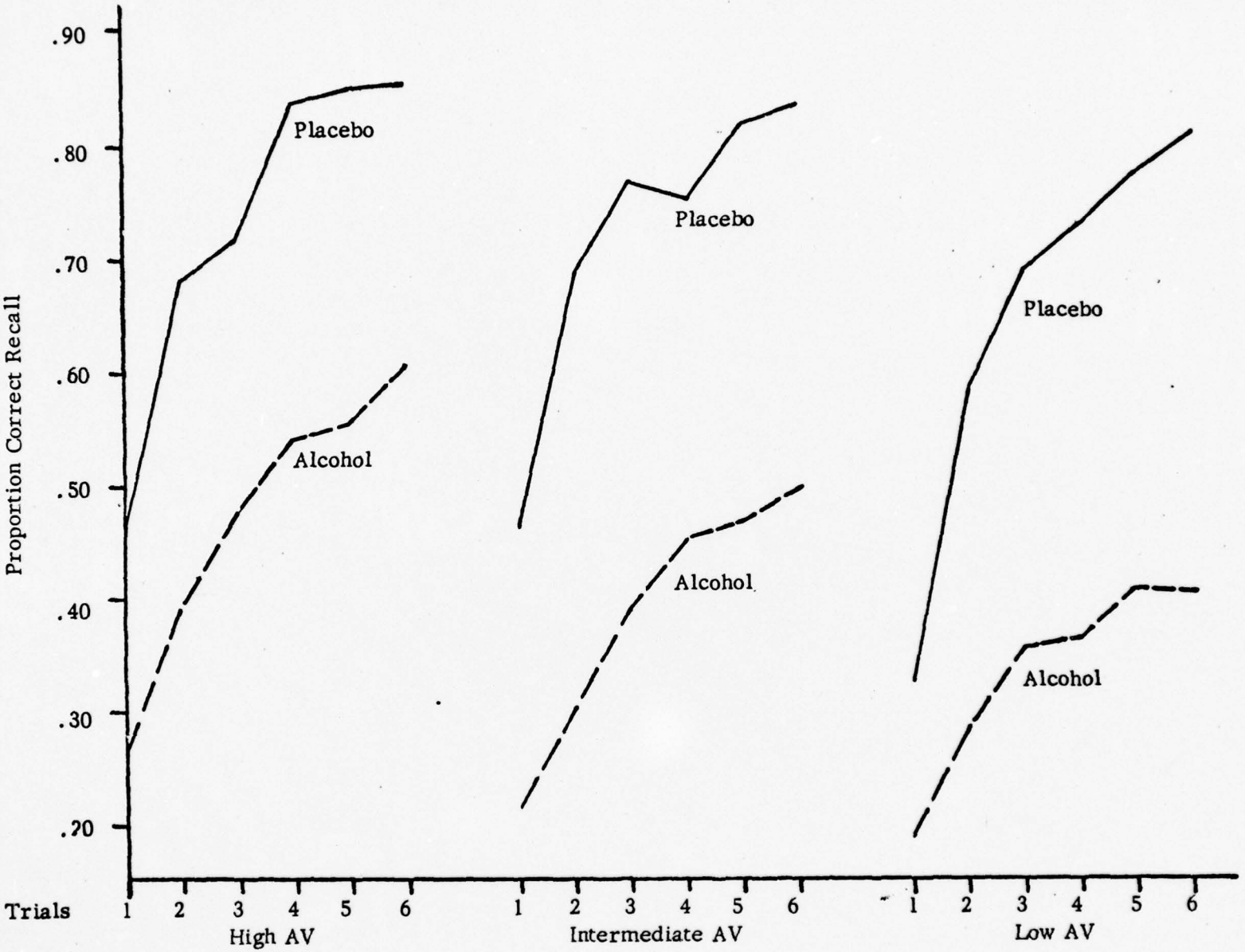

Fig. 12. Three-way interaction of drug, trials, and association value (AV) on recall of associated word-pairs. Simple interactions of drug and trials were significant for intermediate ($p < .05$) and low ($p < .001$) levels of association value.

Analysis of "clustering" in recall (i.e., the recall of paired associated as pairs) is currently in progress. So far, the data indicate that both alcohol and association value impair the recall of word-pairs per se.

In summary, alcohol impaired functions related to both STS and LTS. The STS impairment may be simply a decreased storage capacity; although, other interpretations are possible (e.g., impaired retrieval). The LTS effect appears to be a function of impaired organization and retrieval. Moreover, it is probable that STS impairment leads to the entry of fewer items into LTS. Thus, alcohol may have two effects on LTS: (1) registration of fewer items because of an impaired STS, and (2) less efficient organization and retrieval of those items which are transferred to LTS.

2. Effects of Barbiturates on STS and LTS. A pilot study (N = 9) using large oral doses (200 mg/70 kg) of butibarbital sodium or pentobarbital sodium has been completed. This study partially replicated Experiment 1 (above) and employed the task variables, list length and filled or unfilled 15-second delay interval, in the free recall paradigm. Main effects of serial position, list length, and interpolated task were significant at the 0.001 level. The barbiturates tended to reduce recall at all serial positions ($F_{1,8} = 4.78$, $p < .10$), and a separate analysis of recall of the last two serial position words (unfilled delay) confirmed the impairment of STS ($p < .05$, t-test). Thus, like alcohol, the barbiturates appear to impair both STS and LTS. Unlike alcohol, however, the barbiturates did not interact with list length ($F = 0.2$), suggesting different loci if impairment for alcohol and the hypnotic-sedatives (i.e., barbiturates may not affect LTS organization and retrieval mechanisms). This conclusion is compatible with the results of the information processing experiments reported in section B, above.

II. Effects of Alcohol on Bioelectric Patterns During Sleep

During this year, the physiological study of sleep profiles in a group of 20 chronic alcoholics and 20 control subjects (Ss) individually matched on age has been extended, and the analysis has been completed. An analysis of the effects of alcohol on sleep in chronic alcoholics has also been completed. These current results were reported at the Texas Research Institute of Mental Sciences Seventh Annual Symposium: "Behavior and Brain Electrical Activity". This paper (Alcohol and Sleep in the Chronic Alcoholic) is to be published by the Plenum Press with the Proceedings of the Symposium. A summary of the more pertinent findings are detailed below.

A. Effects of Alcoholism on Physiological Sleep Patterns

Completed analyses of the EEG sleep data from alcoholic and control Ss confirmed that the amount of stage 4 sleep was significantly reduced in younger alcoholics as compared to age-matched controls, but not in older alcoholics (Table 1). These trends resulted in a significant interaction between the effects of alcoholism and age. The Pearson correlations of age and percent stage 4 were -.59 ($p < .01$) in the controls and -.05(N.S.) in the alcoholics. None of the other stages of sleep showed systematic age effects, but older controls were awake significantly longer than younger controls. The amounts of stage 4 were lower, on the average, in both alcoholics and controls than those usually reported by others for comparable age group. Since we scored

Table 1

Stage of Sleep by Age

		Younger Subjects (Age 24-39)		Older Subjects (Age 40-56)	
		Controls**	Alcoholics**	Controls	Alcoholics
Awake*	$\bar{X}$	7.8	9.3	13.7	11.0
	s	4.0	5.2	9.0	5.4
Stage 1	$\bar{X}$	5.2	10.2	5.6	10.7
	s	2.4	3.6	3.5	4.7
Stage 2	$\bar{X}$	60.9	57.8	62.8	58.3
	s	9.2	3.3	6.9	6.4
Stage 3	$\bar{X}$	9.4	4.9	9.4	3.5
	s	3.5	3.3	4.8	5.3
Stage 4	$\bar{X}$	4.3	0.6	1.2	1.6
	s	4.1	1.1	2.9	3.5
REM	$\bar{X}$	20.2	26.4	20.9	25.9
	s	5.7	3.5	2.5	3.1

* Means and standard deviations for awake are based on percent of total bedtime. All other entries are percents of total sleep excluding waking.

**Means in each column are based on N = 10.

the sleep EEGs by the use of rather rigorous amplitude criteria specified for stage 4 in the Rechtschaffen-Kales manual (1968), this led to the interesting question: Do the low amounts of stage 4 found both with aging and with chronic alcoholism result from loss of frequencies in the delta band (0.5 to 4 Hz), or of reduced amplitude of delta activity, or both?

A major finding during the year was that the disparity between the amount of slow-wave sleep in alcoholic and control Ss is accounted for by a decrease in amplitude of delta activity, while the amount of delta activity as measured by frequency is essentially the same for the alcoholic and control Ss. Average delta periodicity was obtained with a modified Biodata period analyzer (Burch and Childers, 1963) which coded the EEG signal into square wave trains and classified their periods into frequency bands. The Delta Index, expressed as the percent of time delta activity was present, was averaged over 5-minute epochs. Amplitude data were obtained over the same 5-minute epochs by a reset integrator and are expressed as mean microvolts (peak to peak).

Figure 13 illustrates the results of independent quantitative analysis of data periodicity and EEG amplitude over the first six hours of the night in a 27-year-old alcoholic and his age-matched control. Both subjects show two well-defined peaks in density of delta periodic activity (Delta Index) shortly after the first and second hours of sleep. However, for the alcoholic patient, the average EEG amplitude function is markedly depressed at these same peaks of delta frequency activity.

Figure 14 shows similar time functions for a 52-year-old alcoholic and his matched control. Again, the alcoholic's EEG amplitude function appears markedly depressed relative to the control subject at the peaks of delta periodicity. At this writing, quantitative EEG analyses are not yet completed for all subjects, so that the results shown above must be treated cautiously. However, these trends do suggest that alcoholic patients may generate sufficient activity in the delta frequency band to qualify as stage 4 sleep, but insufficient voltage to reach the Rechtschaffen-Kales (1968) criterion for that classification. There is nothing in the analyses illustrated in Figures 13 and 14 to indicate whether aging differentially affects the two parameters. These preliminary results suggest that visual classification of the sleep EEG by an experienced scorer is particularly sensitive to amplitude changes and further suggest that different mechanisms are responsible for the amplitude and slow-wave characteristics of the EEG during sleep. There is reason to suppose that, whereas the pacemakers for various brain rhythms are located in subcortical structures, their ultimate voltage depends on the integrity of the cortex. For example, Jouvet (1961) showed that in cats an intact cerebrum was necessary for slow-wave sleep to occur, and Kogan (1969) found evidence in cats that the initiation of high-voltage sleep waves is an excitatory process which originates in deep layers of the frontal cortex.

As reported last year, alcoholics had three times as many brief arousals during the night ($p < .001$) as controls, as well as more frequent changes of stage ($p < .01$) and longer latencies to sleep. Our data in chronic alcoholics indicate that the rhythmicity of the REM cycle is accelerated and that REM periods are disrupted in chronic alcoholic Ss. Figure 15 shows the average distribution of time intervals from REM offset to REM onset for alcoholics and controls on baseline nights. Note that in each group the distribution is bimodal. With time intervals on the abscissa, the modes to the left of 12 minutes represent the proportions of intervals which we classified as REM disruptions, whereas the modes to the right (between 60 and 90 minutes) represent the basic period of the REM-to-REM cycle. The mode for that cycle is about 15 minutes shorter in

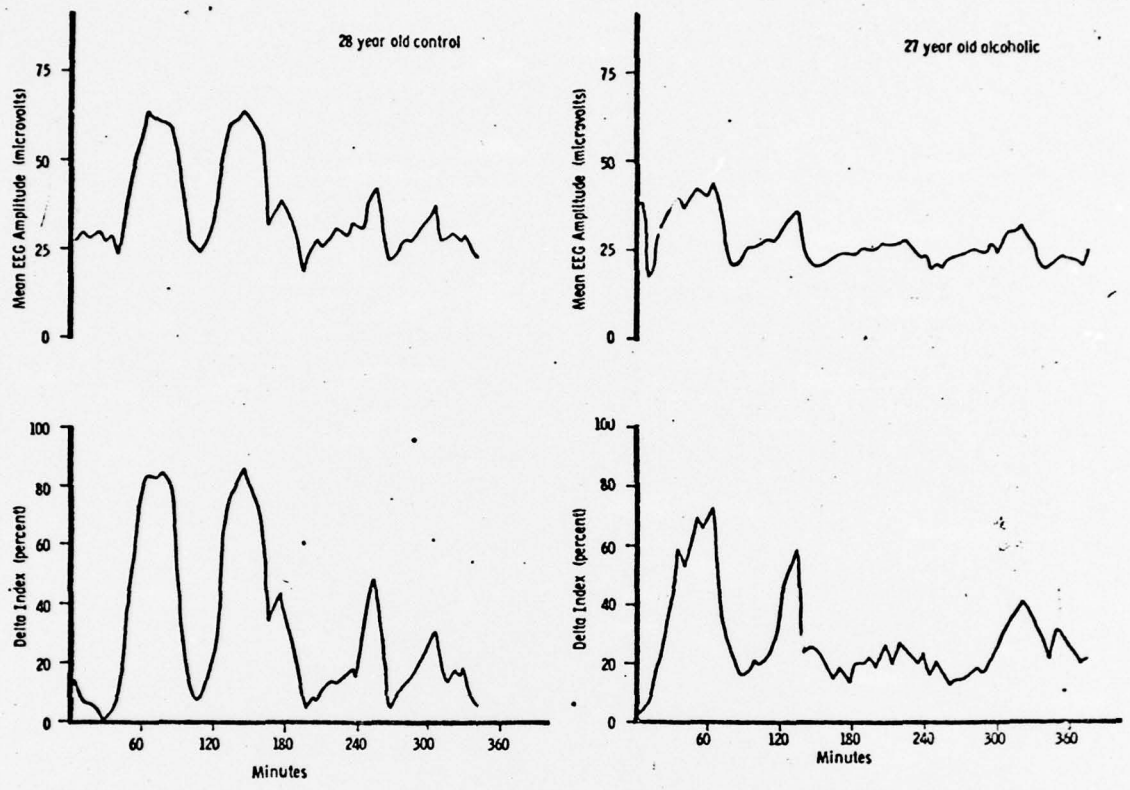

Figure 13. Periodicity of EEG Amplitude and Delta Index during sleep. The charts on the right represent one night of sleep for a sober, younger alcoholic, and the charts on the left represent his matched control S. Delta Index expresses the percent of the EEG record occupied by 0.5 to 4 H_z activity. Note the relative depression of the EEG amplitude peaks in the alcoholic.

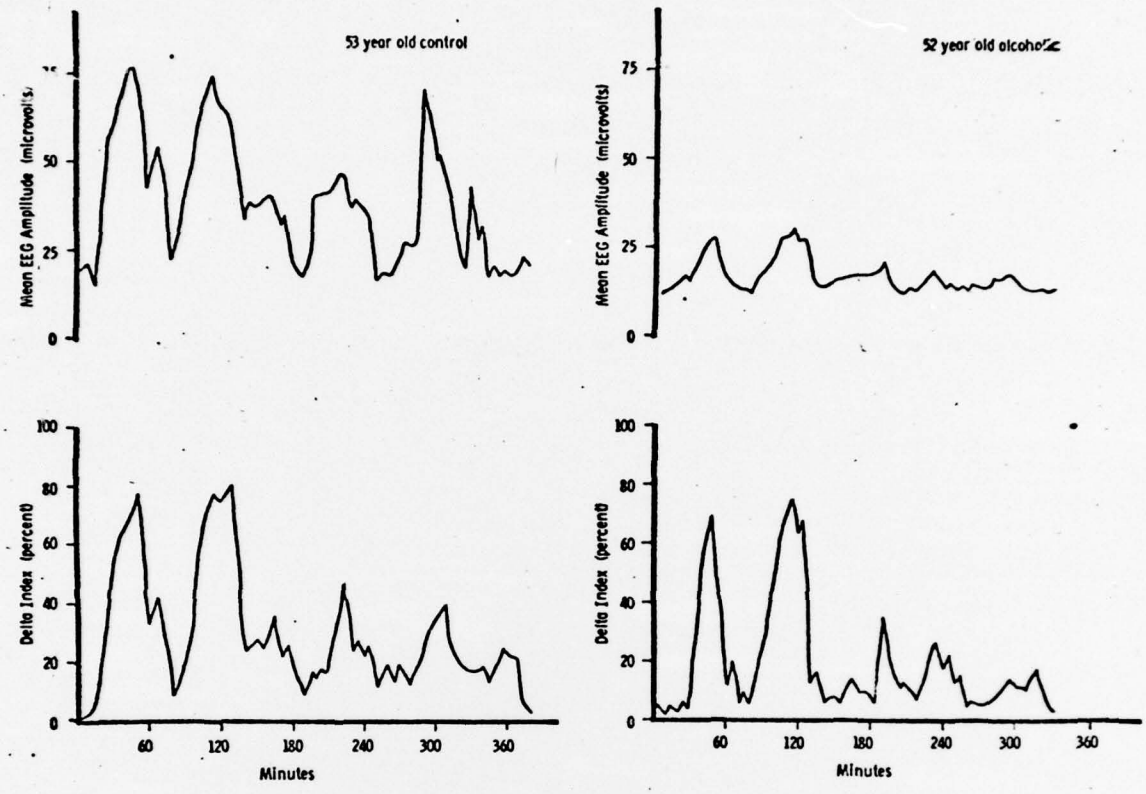

Figure 14. Periodicity of EEG Amplitude and Delta Index during sleep. The charts on the right represent one night of sleep for a sober, older alcoholic, and those on the left represent his matched control. Delta Index expresses the percent of the EEG record occupied by 0.5 to 4 H_Z activity. Note the relative depression of the EEG amplitude peaks in the alcoholic.

the alcoholics than in the controls, and the difference between corresponding means is statistically significant. The latency of onset of the first REM episode was also significantly shorter in alcoholics than in controls, averaging 64 minutes (S.D. = 4.2) and 102 minutes (S.D. = 6.1) in alcoholics and controls respectively. Thus, as indicated, the increased amounts of stage REM found in alcoholics were due to accelerated REM periodicity rather than increased REM period duration. The modes on the left in Figure 15 also illustrate the considerably higher proportion of REM disruptions in alcoholics than in controls, a difference which is significant beyond the .001 level.

Another periodic function associated with sleep is the spontaneous galvanic skin responses (GSRs) which are primarily associated with slow-wave sleep. In the past, we (Lester, Burch & Dossett, 1967) have assumed that spontaneous GSRs during sleep might be a function of both slow-wave sleep activity and of pre-sleep stress. Our findings during this year show that alcoholics have higher rates per minute of non-specific GSRs than controls during waking and in all stages of sleep ($p < .01$). As Table 2 indicates, in alcoholics the GSR rate during stage REM (3.6/min.) approached the waking rate, an unusual finding. Spontaneous GSRs are rare during stage REM in normal subjects (e.g., Johnson and Lubin, 1966). The overall discharge rate for GSRs in our controls was similar to that reported by Johnson and Lubin for normal subjects, but the average rates for alcoholics were about three times higher than normal values.

Average heart rates per minute were not different for alcoholics and controls, but average respiration rates were higher for alcoholics in all stages of sleep and during waking, the differences between means being statistically significant for awake and stage 2. Rates per minute of sigma spindles in stage 2 and rapid eye movements in stage REM were not different for alcoholics and controls.

B. Effects of Intoxication on the Sleep Patterns of Alcoholics

During the two days of drinking by the alcoholic patients, hourly BACs during the first day ranged from 130 to 180 mg percent (mean = 157); for the second day, the range was 115 to 190 mg percent (mean = 159). On day 1, BACs just prior to bedtime ranged from 110 to 165 mg percent (mean = 145), and on day 2, bedtime values ranged from 110 to 190 mg percent (mean = 155).

The effects of alcohol on the EEG stages of sleep were most striking in the first half of the night. For example, alcohol significantly suppressed stage REM and stage 1 and reduced waking time, while potentiating stage 4 in the first half of both alcohol nights (A_1 and A_2). Table 3 shows these effects. Stage 3 also increased during the first half of each alcohol night, the change being significant ($p < .01$) on A_1 but not quite significant on A_2 ($p < .10$). Percent awake was further reduced in the first half of A_2 relative to A_1, but there were no other significant differences between A_1 and A_2. Alcohol effects for the second halves of A_1 and A_2 were not systematic. Alcohol also decreased the number of brief arousals, changes of stage of sleep, and the number of REM disruptions in alcoholics, all measures which are increased in alcoholics as compared to controls.

In Table 4 where all-night stage of sleep scores are categorized by age, it can be seen that the relative decreases caused by alcohol in waking, stage 1 and REM were similar for both age groups, as was the increase in stage 3. These changes were statistically significant for both younger and older alcoholics on both A_1 and A_2. In the younger group, alcohol also caused a considerable and significant increase in percent stage 4, from about 0.6% on baseline nights to 7.1% on A_1 and 6.1% on A_2 ($p < .01$). However, in the older subjects, the small increase in stage 4 was not significant on either of the alcohol sessions. These differential increases in the two age groups resulted in a significant interaction between the effects of alcohol and age on percent stage 4. Thus, alcohol poten-

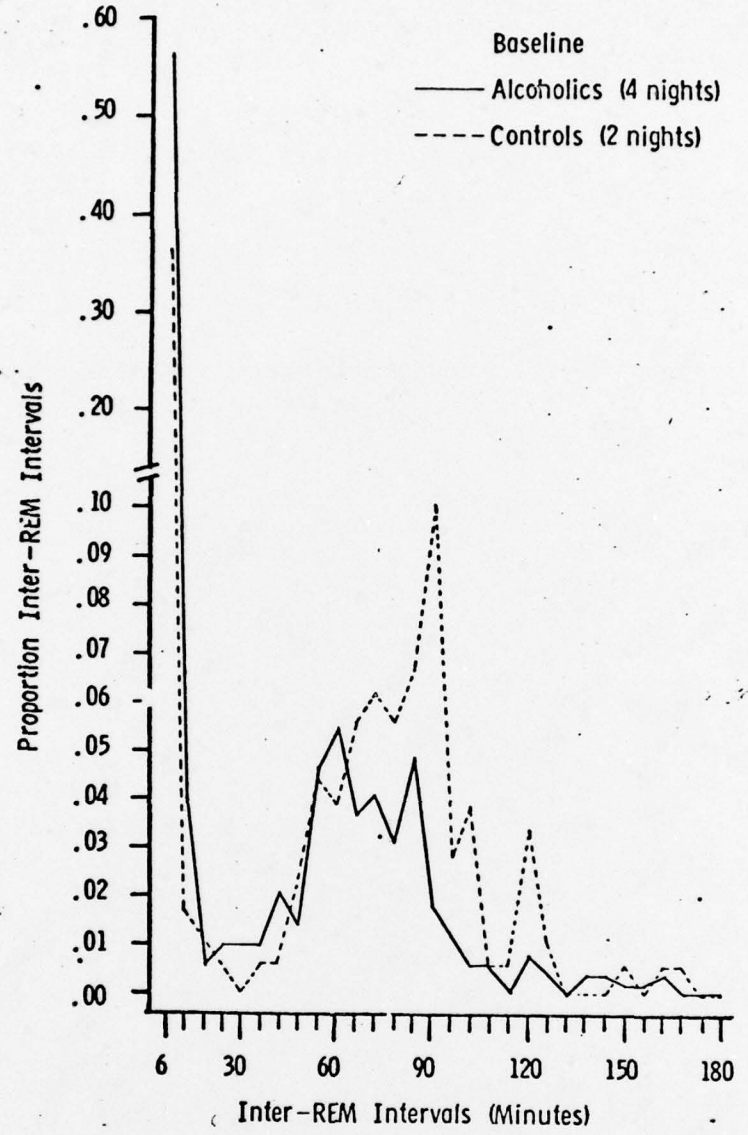

Figure 15. Proportional Distribution of Inter-REM Intervals in 20 Chronic Alcoholics and Age-Matched Controls. Note the compression of scale in upper half of ordinate.

Table 2

Autonomic and Phasic Variables

Controls

		GSR Rate (per Min.)	Respiration Rate (per Min.)	Heart Rate (per Min.)	Spindles in Stage 2 (per Min.)	Eye Movement* Density in REM
Awake	$\bar{X}$	1.5	14.0	69.9		
	s	1.2	2.3	6.5		
Stage 2	$\bar{X}$	1.8	15.7	61.6	3.0	
	s	2.0	2.4	6.1	2.4	
Slow Wave	$\bar{X}$	3.5	16.3	62.7		
	s	3.1	2.6	6.1		
REM	$\bar{X}$	0.7	16.6	65.7		3.9
	s	0.8	2.3	5.0		0.7
All Stages	$\bar{X}$	1.9	15.6	65.0		
	s	2.2	2.6	6.8		

Alcoholics

		GSR Rate (per Min.)	Respiration Rate (per Min.)	Heart Rate (per Min.)	Spindles in Stage 2 (per Min.)	Eye Movement Density in REM
Awake	$\bar{X}$	5.1	17.5	69.7		
	s	2.8	3.5	5.0		
Stage 2	$\bar{X}$	5.8	19.1	66.3	3.0	
	s	3.0	4.9	5.0	2.1	
Slow Wave	$\bar{X}$	9.4	18.0	64.5		
	s	2.9	3.2	6.8		
REM	$\bar{X}$	3.6	18.6	65.4		4.4
	s	2.7	4.0	4.5		1.0
All Stages	$\bar{X}$	5.5	18.4	66.8		
	s	3.4	4.1	5.5		

*Average number of ten-sec. epochs per min. containing rapid eye movements.

Table 3

Alcohol and Stages of Sleep in Alcoholics

		1st Half of Night				2nd Half of Night			
		Baseline	Alcohol 1	Alcohol 2	Recovery	Baseline	Alcohol 1	Alcohol 2	Recovery
Awake*	$\bar{X}$	13.3	5.3	3.4	16.7	6.9	5.8	7.5	11.3
	s	8.5	5.6	4.0	16.6	6.7	8.7	9.3	19.4
Stage 1	$\bar{X}$	11.2	5.1	4.2	8.2	10.2	8.2	9.7	9.2
	s	5.6	4.4	2.9	5.3	4.4	5.7	5.2	8.5
Stage 2	$\bar{X}$	60.4	60.9	62.6	58.7	55.7	60.2	57.2	53.3
	s	9.3	17.9	13.3	10.0	5.0	9.3	6.8	9.9
Stage 3	$\bar{X}$	7.8	13.0	11.8	14.3	0.8	1.1	0.5	3.5
	s	7.6	10.4	10.5	9.8	2.2	2.8	1.6	5.6
Stage 4	$\bar{X}$	2.2	10.2	8.0	1.5	0.1	0.0	0.0	0.1
	s	4.9	13.5	10.9	3.0	0.4	0.0	0.1	0.3
REM	$\bar{X}$	18.4	10.7	13.3	17.3	33.1	30.5	32.5	33.9
	s	3.8	4.1	6.4	6.0	4.6	7.6	7.3	7.5

* Means and standard deviations for awake are based on percent of total bedtime. All other entries are percents of total sleep with waking excluded.

Table 4

Stage of Sleep by Age in Alcoholics

		Younger Subjects** (Age 24-39)				Older Subjects (Age 40-56)			
		Baseline	Alcohol 1	Alcohol 2	Recovery	Baseline	Alcohol 1	Alcohol 2	Recovery
Awake*	$\bar{X}$	9.3	4.1	4.3	10.7	11.0	7.2	6.4	16.5
	s	5.2	3.5	3.8	9.3	5.4	6.7	6.5	13.1
Stage 1	$\bar{X}$	10.2	5.5	6.0	8.1	10.7	7.6	7.2	8.5
	s	3.6	2.7	3.3	4.9	4.7	5.1	2.9	7.4
Stage 2	$\bar{X}$	57.8	57.4	59.1	58.3	58.3	63.3	61.5	54.4
	s	3.3	6.6	6.4	6.8	6.4	11.8	7.7	7.0
Stage 3	$\bar{X}$	4.9	8.4	7.0	8.5	3.5	6.3	6.8	9.3
	s	3.3	4.9	4.7	6.8	5.3	7.7	8.7	5.1
Stage 4	$\bar{X}$	0.6	7.1	6.1	0.1	1.6	3.5	2.6	1.6
	s	1.1	7.7	6.6	0.2	3.5	5.9	4.2	1.9
REM	$\bar{X}$	26.4	21.5	21.8	25.0	25.9	19.2	21.8	26.2
	s	3.5	4.9	5.3	6.2	3.1	4.3	8.2	3.4

* Means and standard deviations for awake are based on percent of total bedtime. All other entries are percents of total sleep with waking excluded.

**Means in each column are based on N = 10.

tiated stage 4 sleep in the younger but not in the older alcoholics. This is interesting and perhaps important. Gross et al. (1973) found similar, but transient increases in slow-wave sleep in alcoholics in their thirties who drank heavily for four to six days. The data indicate that in the young alcoholic, cerebral mechanisms for the generation of high-voltage delta activity can function if stimulated pharmacologically. Whether the failure to induce stage 4 in the older patients results from prolonged abuse of alcohol or normal aging or both will not be understood until older normals are given alcohol. In a study in progress at the present time, alcohol did potentiate slow-wave sleep in two of the three volunteer Ss who were in their late forties.

The validity of notions concerning premature aging or permanent brain damage in the chronic alcoholic depends partly on a demonstration that the disordered sleep profile does not recover with prolonged sobriety. Our data on this issue are not sufficient, but they do indicate that over a period of at least five weeks of sobriety, the abnormal features of sleep in alcoholics remained rather stable. Nevertheless, the increase in stage 3, and nonsignificant trends toward improved quality of sleep by the final recovery session, encourage the hope that, given sufficient time, the sleep profile of the sober alcoholic may recover to age-appropriate norms.

C. Effects of Alcohol on Autonomic and Phasic Variables in Alcoholics

Alcohol reduced the average frequency per minute of non-specific electrodermal responses in the first half of the first alcohol night (A_1). These effects were statistically significant for stages 2, SW and REM, and borderline ($p < .10$) for awake. On A_2, the reduced GSRs continued as trends ($p < .10$) except for stage SW where the reduction maintained significance ($p < .01$). Average heart rates which had not been different for sober alcoholic patients and matched controls were systematically increased by alcohol in alcholics, throughout each alcohol session and in all states of consciousness. Alcohol had no systematic effect on rate of respiration. The frequency per minute (density) of sigma spindles in stage 2 decreased significantly on both halves of A_1 and A_2, but the density of rapid eye movements was reduced only on A_1. None of the alcohol effects on phasic and autonomic variables were related to age.

Except for respiration, the pattern of changes found in these autonomic and phasic variables is qualitatively similar (though more systematic from session to session) to that described by Rundell et al. (1972) in young normals, where alcohol also increased heart rate throughout the night and tended to decrease the density of such phasic events as GSRs, sigma spindles in stage 2 and rapid eye movements in stage REM. Whereas the present study showed no alcohol effects on respiratory rates, Rundell et al. (1972) found an increase over three nights of alcohol ingestion.

Our findings indicate that chronic abuse of alcohol is associated with disruptions of the sequential properties of sleep and alterations of some of the periodic functions of sleep profiles. Chronic alcoholism is associated with acceleration of REM sleep periodicity, disruption of sleep with brief arousals, disruption of REM sleep epochs, markedly enhanced GSRs in all stages of sleep, and dissociation of delta frequency amplitude responses. From these data, it seems that a change in strategy might enhance the information obtained from EEG (and other physiological) variables during sleep. This change in strategy would be, in addition to classifying the EEG by the classical sleep stage method, to evaluate the effects of alcohol, chronic alcoholism and age on the periodic (ultradian) oscillations of EEG frequencies during sleep, with particular emphasis on phase relations. An important part of our plans for this year is the evaluation of our existing data base from alcoholic and age-matched normal Ss in both alcoholized and dry states for these functions.

During this year, we expect to complete the study of the effects of alcohol on physiological sleep profiles in volunteer Ss over the age of 45. The purpose of this study is to continue the evaluation of the hypothesis that chronic alcoholism produces changes in sleep profiles resembling premature aging. As noted in the progress report, young alcoholics had an increase in slow-wave sleep when alcoholized, while older alcoholics did not. To date, two of three volunteers (ages 45, 46 and 52) had an increase in slow-wave sleep with alcohol. The study of alcoholics who have been drug and alcohol free for two years will also be completed.

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